MEMOIR

ON THE

INFLUENCE OF HYPERTROPHY AND DILATATION

ON

DISEASES OF THE HEART;

AND ON SOME POINTS IN THEIR

DIAGNOSIS AND TREATMENT.

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NOTE.

The following cases and observations appeared in the Edinburgh Monthly Journal of Medicine; and they have been thrown off in a separate form, with few alterations, and these chiefly verbal. They were brought forward simply as clinical illustrations of certain points which have not attracted due attention; and regarding some of which prevailing notions are not sufficiently exact.

In some of the sections there are recorded cases of more than ordinary interest; for example, in Section II., and Cases XIV. and XV.

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DISEASES OF THE HEART.

CASES AND OBSERVATIONS.

SECTION I.

HYPERTROPHY AND DILATATION.

HYPERTROPHY and Dilatation of the Heart have been long and justly regarded as conditions secondary in their nature, and depending on some obstruction to the circulation. They may arise from causes widely different from each other; but the most usual, as well as the most important, are the organic affections of the heart; and few of these exist without more or less hypertrophy or dilatation. The frequency with which hypertrophy and dilatation occur as complications of the organic affections of the heart, confers an almost universal importance on them; and the intimacy of their mutual relation—for the one probably never exists without the other—suggests the necessity of a careful inquiry into their effects and influence.

It was the opinion of M. Bertin, and this has been corroborated by more modern investigations, that hypertrophy, though capable of giving rise to troublesome and even dangerous accidents, is not in itself a dangerous affection.

Dilatation has been esteemed dangerous, chiefly because believed to indicate that obstruction of the circulation has become extreme; and the cause of that obstruction, from which the dilatation arose, has been regarded as the cause of all those secondary evils and distresses which so invariably occur in the last stage of cases of disease of the heart. Now, I think that an error has been committed here, and that the dilatation exerts a material and direct influ-

ence in the development of these secondary evils, while the pre-existing cause of obstruction, whether it be valvular or other, is comparatively unimportant, except in so far as it must be regarded as the origin or commencement of a series of changes which is to result so formidably. In short, while the dilatation is *secondary* in regard to the period of its development, it becomes *primary* in its ultimate importance, acting as the *chief* cause of all the most dreaded evils of the diseases of the heart.

The intimacy of the relation which subsists between hypertrophy and dilatation of the heart creates some difficulty in the inquiry, and has occasioned not a little confusion in the attempt to indicate the distinctive signs of these two forms of disease. Without entering into the details of the discussion, it may be affirmed that one of these conditions does not exist in any case without the other, or very rarely; and that, in consequence of this co-existence of hypertrophy and dilatation, the effects which would arise from one or other of these alterations of the heart are materially modified; they are not, however, to be regarded as antagonistic of each other; therefore the one does not necessarily correct the evil effects of the other. Nevertheless, as hypertrophy involves increased *power*, and dilatation increased *capacity*, in so far as the latter induces any diminished power, its evil effects may be counterbalanced and delayed by the hypertrophy. In this want of direct antagonism between these two conditions, we have an explanation of the fact, that no amount of muscular hypertrophy is inconsistent with dilatation of the heart and all its most formidable effects, for we see these arising in cases in which the heart has acquired three or four times the normal amount of muscular substance. This fact suggests the question, how far the embarrassment of the heart's action may be due to a disorganised state of the muscular substance? but this inquiry is beside the object of the present memoir, and will not be entered upon.

The leading object of the following reports is to show the supreme importance of hypertrophy and dilatation, especially the latter, in the diseases of the heart; and also to point out their frequent, if not their invariable co-existence. These propositions will be demonstrated from the facts of almost every form of heart disease; and, at the same time, the details of the cases will afford many opportunities of considering other questions of practical importance.

In the course of the reports and observations, the degree of hypertrophy and dilatation will be of necessity frequently alluded to. It is impossible to convey a precise notion of the state of the heart in regard to these two conditions, and the relative proportion they bear to each other, because of the indefiniteness of the terms which must be employed in their description. An approximation only can be made to accuracy, by stating the weight along with the general bulk and capacity of the organ. But, after all, the description of the degree of hypertrophy or dilatation resolves itself very much into an expression of opinion, that this or that was the state of the organ.

CASE I.—*Dilatation of the Heart; Hypertrophy; Obstructed Circulation; Chronic Chest Symptoms; Cardiac Symptoms recent; Progress rapid; Death unexpected.*

Alexander Mackay, æt. 50, a coach driver, of robust frame, was admitted into the Royal Infirmary on the 27th November 1847. At the time of admission he had very urgent dyspnœa and tumultuous action of the heart; frequent harrassing cough, with scanty expectoration of mucus tinged with blood. He had the puffy and livid aspect characteristic of obstructed circulation; there was faint jaundice, and slight general œdema. The pulse was so small, rapid, and irregular, that it could not be reckoned. Urine was copious, high coloured, and turbid. The bowels were slow.

History.—He ascribed his illness to exposure to cold and wet, eight weeks previous to admission. His symptoms had been distressing for only four weeks —the dyspnœa and palpitation especially so, and profuse hæmoptysis had occurred. He had not suffered from any definite pain. After careful questioning, he admitted that he had been liable to cough from boyhood, and at times to slight difficulty of breathing—but never to such a degree as to lay him aside from work. He always was much exposed to the weather. He never had rheumatism nor palpitation, till the existing attack. He said his habits were not intemperate.

Physical Signs.—The impulse of the heart was feeble and undefined; the precise situation of the apex could not be determined. As already stated, the action of the heart was tumultuous. The sounds were faint and obscured by cooing and sibilant respiration, which was intense and general. No abnormal murmur accompanied the heart's sounds, but they were irregular.

The hepatic dulness was extended, and the dull space was tender, as was also the epigastrium.

Subsequent Progress and Treatment.—He was leeched in the scrobiculus cordis, and purged with medicine, with immediate and marked relief. He slept quietly; the tumult of the heart's action subsided; the pulse was reckoned readily, 108, synchronous with the heart's beat, and having an occasional irregularity corresponding to the remaining irregularity of the heart's action. He was blistered on the chest, and the urgency of the symptoms continued to decline, with the exception of the jaundice, which became progressively more decided. At the same time there was gradually developed a state of languor and depression; and on the third of December perfect prostration supervened, with tumultuous action of the heart, a fluttering pulse, &c. This state lasted only a few hours, when he died.

Post-mortem Examination.—The surface had a universal bright yellow colour. The volume of flesh was large. There was dropsy of the cellular tissue and cavities.

The heart was increased in its general bulk, and weighed sixteen ounces. It had the rounded form characteristic of dilatation. The left ventricle was distended by a soft recent coagulum, and was manifestly dilated. The thickness of the muscular walls was not very evidently increased at any given point. The right ventricle was dilated, not distended.

The aorta was very little dilated.

Both lungs contained masses of apoplectic condensation.

The liver and kidneys were loaded with blood.

Remarks.—This case is one of a large class which establishes the fact that all the effects of dilatation may arise in cases in which the heart is truly hypertrophied.

The phenomena indicating the dilatation were in no respect different from what have long been regarded as the signs of that state.

Some of these signs, however, I am disposed to think, have a more specific relation to the dilatation than has been usually acknowledged.

Such conditions as lividity, dropsy, hæmoptysis, pulmonary condensation from hemorrhage, and the distended state of the left ventricle, have been very absolutely associated with disease of the heart; but they have not been so pointedly associated with dilatation, as this case and many others testify is the fact. No doubt these conditions occur in other cases than mere dilatation; but even in such cases, a very intimate relation may be traced to dilatation, or to a state of the organ causing the same obstructive effects on the circulation. In the present case, the degree of dilatation was great, and no other lesion existed, unless hypertrophy, to account for the extreme degree of the conditions above referred to; and it will be found that these do not ordinarily exist without more or less dilatation. These symptoms in most cases exist late in the attack, and may be regarded as the index of a preponderating and formidable dilatation.

The post-mortem distension of the left ventricle in this case may be noticed. In the diseases of the heart I have never met with this, except in cases in which the capacity was increased, and the power of the organ consequently embarrassed. In such cases death is usually sudden, or rapid as in this man.

From the facts stated, and from the physical signs in the case, it is manifest that a very embarrassed and enfeebled state of the heart's action existed, but these are the very opposite of the conditions which exist when hypertrophy is in excess, which involves augmented power. We are therefore shut up to the conclusion that the embarrassment of the circulation, and its consequent effects, were due to the dilatation of the heart, notwithstanding co-existing hypertrophy, or increased muscular substance.

It is very difficult to determine what value we shall attach to the statements given by patients of the history of their diseases. In so far as the statement can be trusted in the present case, it is important to observe that his very old-standing chest symptoms were not of heart disease, but probably of a chronic bronchitis. This state of the lungs, by hindering the freedom of the circulation, was no doubt the primary cause of the cardiac changes which gradually and insidiously supervened, and only manifested themselves when, from accidental causes, the balance of the circulation was more disturbed; and then, the defective state of the heart incapacitated it for the work of restoring the equilibrium of the circulation; and the subsequent rapid progress of the case, with prominent *heart* symptoms, became remarkable. This rapid progress in the later stages is to be taken in connection with the very dilated state of the heart.

In the treatment of this case we see a well marked instance of the benefit of moderate leeching to the scrobiculus cordis. The ultimately unfavourable result of the case, with recurrence of all the

worst effects of dilatation, only renders the temporary but decided relief afforded by leeching the more remarkable.

CASE II.—*Hypertrophy of the Heart; Pleuro-Pneumonia: Sudden Death.*

John Craig, æt. forty-nine, a blacksmith of large frame, but spare, and according to his own statement, of temperate habits, was admitted on the 19th May 1845, with urgent dyspnœa, cough, and expectoration, and œdema of the lower extremities and abdomen.

History.—He stated that these symptoms had existed more or less for three months, accompanied by attacks of palpitation; and that up to this time the state of his health had never attracted his attention.

Physical Signs.—The pulse was hurried, visible, synchronous with the cardiac impulse, but irregular. The cardiac impulse was diffused—communicated to the upper part of the belly; it was irregular in force as well as in frequency. The apex beat in the sixth intercostal space, five inches left of the mesial line. The præcordial dulness did not exceed two inches in vertical diameter, and was bounded beneath by tympanitic percussion, which existed higher in the left front than is usual. The horizontal diameter extended from the mesial line, five inches leftwards,—the dulness was incomplete in the outer three inches. The heart-sounds were obscured by intense bronchitic cooing and sibilance.

The right side of the chest was dull on percussion, except in the subclavian space, with enfeebled respiratory and vocal signs.

Progress.—On the evening of the 20th, after rising from bed, he died suddenly.

No treatment of importance had been put in practice.

Post-mortem Examination.—The right pleura contained a large quantity of turbid serum, and lymph in loose flakes, and layers adhering to both pleural surfaces. The middle and inferior lobes of this lung were condensed in the second stage of pneumonia.

The heart had the rounded form of dilatation; it weighed 23 oz., and was much increased in general bulk. It was dilated in all its cavities. The valves were competent. The aortic valves were increased in their depth by a supplementary, membranous, translucent formation from the margin of each valve. The comparatively thickened and opaque margins of the original valves were well defined, where the new formation was attached; and to this margin on the ventricular surface there were attached small wart-like excrescences. The other organs were healthy.

Remarks.—This man's employment is to be noted in connection with the degree of hypertrophy of the heart. There is a very intimate connection between the higher degrees of hypertrophy in many cases, and those occupations in which violent and protracted muscular effort is called forth. This is the case, too, when the hypertrophy is secondary to valvular disease.

There is reason to believe that we were well informed, that cardiac symptoms had made their appearance only within three months. But it cannot be affirmed how far these symptoms depended upon the state of the heart: or how far they may have been influenced by the state of the right lung and pleura. In so far as they depended upon the altered state of the heart, the hypertrophy cannot be supposed to have been in any great degree their cause;—that hypertrophy certainly existed, during a long period antecedent to the

date of the symptoms, when, indeed, his health was thought good. The origin and progress of the dilatation cannot be determined—it, too, may be supposed to have existed long previously, without having caused any embarrassment of the circulation; and the pleurisy may be considered the active agent, exciting the symptoms of his last illness. Be this as it may, the symptoms were mainly those of an embarrassed circulation, which neither pleurisy nor hypertrophy of the heart can of themselves explain; hence, we infer the importance of dilatation in influencing the progress of such cases. Cases, illustrative of this point are not rare; and while any organic disease modifies the progress of an accidentally developed acute inflammation, there can be no doubt that advanced dilatation of the heart complicates such a case most seriously—embarrassing the treatment—and exposing the patient to the most imminent risk. Hypertrophy of the heart, on the other hand, when dilatation is slight or wanting, is in no degree inconsistent with speedy and complete recovery from an acute attack.

Sudden death in cardiac diseases is connected, in a large proportion of cases, with dilatation;—though, as in this man, the dilatation may co-exist with extreme hypertrophy. Such a mode of death is one of the chief risks in all cases in which dilatation of the heart has an acute inflammation superadded to it.

The facts of this case afford very direct evidence of the value of certain physical signs. The pulse had neither the small size nor indistinctness which frequently accompanies dilatation, owing, probably, to the muscular hypertrophy of the heart.

Another condition of the pulse to be noted in this case is, that it was visible. In commenting upon other cases, the opportunity will occur of giving a more extended view of the value of this sign. All that can be said now is, that it is noticed chiefly in cases in which, as in this man, a large hypertrophy is accompanied by an increased capacity of the heart. This cannot be regarded as of itself an important sign; it occurs in many cases in which no embarrassment of the heart's action exists; and, from this fact, I am disposed to connect it more directly with hypertrophy than with the dilatation of the organ.

The impulse of the heart affords important data for determining the degree of dilatation. It is manifest that such hypertrophy as existed in this man, must greatly modify the impulse, which usually becomes *extended*, and more or less *forcible*. Now, from the relative degree of these two conditions, the degree of dilatation may be, in in some measure, determined. As the dilatation increases, the force becomes less in proportion to the extent; but still more striking will the contrast appear between the *feebleness* or undefinedness of the impulse, and the *extent* of the præcordial space dull on percussion.

The extended horizontal diameter of the præcordial space of dull percussion, is an important indication of dilatation. It cannot be affirmed to be of itself absolutely significant of dilatation, as will be

illustrated in other cases, but in connection with other signs, especially the state of the impulse, it is the most invariable and important of the physical signs. It bears a very direct relation to the rounded form of the heart which occurs in dilatation.

The displacement of the apex of the heart to the sixth intercostal space is a sign, as to whose value I am not confident. It has been regarded as very absolutely indicative of hypertrophy; and I have not met with it, except in such cases. But it frequently does not occur; and the apex still presents in the fifth space, in many cases of very excessive hypertrophy—even when the hypertrophy is of the left ventricle mainly; but still more certainly where the right ventricle is the seat of the hypertrophy. In the present case, the bulk of the heart was partly due to the hypertrophy, which was general; though it must not be overlooked that the capacity of the cavities was great.

The supplemented state of the sigmoid valves is very unusual. I do not remember to have met with it in the aortic valves before; it is believed to occur more frequently in the pulmonic valves, though even there it is rare. We have no means of ascertaining whether these valves had at any former time been incompetent—they had not the aspect of having ever been sufficiently deformed for this.

The following case was under observation several months. The leading facts only will be reported; without a minute account of its progress and changes:—

Case III.—*Hypertrophy of the Heart; Dilatation; Death from Erysipelas.*

Andrew Ormiston, æt. 44, a waggon-driver, was admitted on the 17th Dec. 1845. He was a robust man, of intemperate habits. He had a bloated countenance, slight general œdema, and urgent dyspnœa. His sleep was disturbed: and violent paroxysms of angina pectoris occurred. He had little cough, and no expectoration.

History.—He stated that these symptoms, with occasional fits of palpitation, had been troublesome only within a fortnight; they occurred suddenly, and without any definite pain. He had been unable for his work for ten days.

Physical Signs.—The heart's action was tumultuous. The impulse was widely diffused, and not proportionately forcible. The præcordial space dull on percussion had a horizontal as well as a vertical diameter of five inches. The sounds of the heart were free of murmur. The first was prolonged, but obscure; and at times reduplicated. The second was increased in loudness, at the base of the heart. The pulse at the wrist was always small, and more or less irregular; it was imperfectly synchronous with the heart's impulse.

The left side of the chest was extensively dull on percussion; with feebleness of the respiratory signs; and some obscure wheezing and sub-mucous rattle.

Progress.—In the progress of the case the symptoms underwent frequent variations. The physical signs continued very much as reported at the time of admission. His great distress resulted from the paroxysms of angina pectoris, with dyspnœa and tumultuous action of the heart; and during those attacks dropsy was usually considerable, and the urine scanty. Angina frequently existed without dropsy; but the most marked and continued relief to this, and to other symptoms, resulted when copious diuresis occurred—whether it was spontaneous or the effect of treatment. Up to the last, though dropsy had nearly disappeared, the paroxysms were severe and frequent. On the 28th

April, four months after the cardiac symptoms had become troublesome, erysipelas of the head occurred, and he died on the 30th.

Treatment.—The principal treatment, and that whose effects were most apparent, consisted in small local and general blood-letting, and the administration of diuretics.

Up to a late date the application of three or four leeches to the scrobiculus cordis, afforded speedy relief to the anguish of the paroxysms; and was at times succeeded by copious diuresis. On one occasion, when the urine was nearly suppressed, dropsy was considerable, with dyspnœa, cough, and the signs of general bronchitis; and a tumultuous state of the heart's action; all treatment having proved unavailing, the remedies were omitted, and digitalis given in the form of infusion. Within twenty-four hours, diuresis commenced; and in a few days the daily quantity averaged 80 oz.,—at the same time the heart's action was calmed; and the pulse fell to 50, with a rare irregularity, and his distress became altogether less. The quantity of the infusion taken was under 3 oz. in the course of thirty hours.

Post-mortem Examination.—I regret this part of my report is very imperfect; the notes of the post-mortem examination having been mislaid. The following statements are made from memory; but may be relied on so far as they go:—

The heart was hypertrophied in all its parts: and dilated in both ventricles,—the left most decidedly, the walls of which were nearly an inch thick. No lesion of the valves existed. The left pleura contained several quarts of high-coloured serum, with much lymph in loose masses, and in membranous layers on both pleural surfaces.

Remarks.—This case contrasts with the others of this series in several particulars. One of the most striking of these is the manner of death, and its immediate cause. In the other cases death resulted more or less directly from the state of the circulation. This man was cut off by erysipelas. It has been asserted that there is a tendency to such a termination in cases of disease of the heart; but I have been quite unable to trace any consistent relation between them. They do not concur more frequently than may be explained by accidental causes.

The physical signs of the dilatation agree only in part with the signs in the other cases. It is important to bear in mind this partial disagreement; it will be observed more or less in every case; and arises from the different degrees of the dilatation and hypertrophy.

In this, as in the other cases, the signs of the greatest value were the horizontal extent of dull percussion, and the disproportionate feebleness of the impulse compared with its extent. The *smallness* and *want* of *synchronism* of the radial pulse with the heart's beat, are valuable signs; and are closely connected with the dilatation; though it may be seen, from other cases of this series, not always to occur even when dilatation is great. The reduplication of one of the heart's sounds is a sign of which I have been unable to discover any satisfactory explanation. It usually affects the second sound; and I have been in the custom of attributing it to mere irregularity of muscular contraction, and consequent want of synchronism in the valvular and tendinous tension on which the heart sounds largely depend. The *muffled* state of the first sound demonstrates, that per

haps too much value has been attached to mere clearness of tone of this sound, as indicative of dilatation. It is true that the muffled tone arises from muscular hypertrophy; while clearness is connected with dilatation. Still the fact in this case must be borne in mind, that the muffled tone of increased muscular substance persisted, notwithstanding the supervention of dilatation and its gravest effects.

There are other points in this case suggestive of important questions. I shall not, however, occupy space in the meantime by taking up these, as the facts do not establish any specific and practical rule. I refer especially to the angina and its relation to the state of the heart; and to the effect of the diuretics, which act most capriciously under all circumstances, in the diseases of the heart, and appear to be much hindered by the obstruction to the circulation arising from dilatation.

Case IV.—This case is necessarily imperfect, a post-mortem examination not having been permitted. The *dilatation* of the heart was, however, so manifestly indicated by the signs, that the case is instructive.

Hannah Henderson, æt. 25, a domestic servant, was admitted into the hospital on the 2d February 1845. She was slender in her general development, and had a slight abnormal prominence of the sternum and costal cartilages. She was labouring under general dropsy, urgent dyspnœa, palpitation, and cough with expectoration; her face was swelled and livid. She also suffered from sickness and vomiting, with acute tenderness of the epigastrium.

History.—She could not give a definite account of the date and origin of these symptoms. She said she never had been strong; and had been liable to distressing paroxysms of dyspnœa and palpitation for years, but never such as to unfit her for work. Her existing extreme distress had occurred within a fortnight, when the swelling had first attracted attention.

Physical Signs.—Mucous and sub-mucous rattle existed in all parts of the chest. The apex of the heart appeared to beat in the fifth intercostal space. The impulse was neither diffused nor forcible. The space of præcordial dull percussion extended five inches leftwards from the mesial line; and from the third left cartilage to the costal margin. The sounds of the heart were hurried, regular, and free of murmur. They presented the clearness of tone which has been considered characteristic of dilatation. The radial pulse was small. The veins of the neck were permanently gorged.

Progress and Treatment.—Small cuppings from the chest were employed, with laxatives and diuretics. This effected a very slight and temporary benefit. The urine was always scanty; the pulse became irregular; dyspnœa and vomiting caused much distress; and latterly the dropsy greatly increased. She fell into a state of semi-stupor, which lasted several days, with gradual exhaustion, and she died on the 19th March.

Remarks.—The steadily unfavourable progress of this case is characteristic of that state of the heart in which the dilatation has disproportionately overbalanced the power, or hypertrophy, of the organ.

The horizontal extent of the præcordial dull percussion, the smallness of the radial pulse, and the clear tone of the first sound of the heart, corroborate each other as indicative of dilatation. It is useless to inquire what was the degree of hypertrophy in the absence of

anatomical evidence. The form of the chest, the vertical extent of præcordial dull percussion, and the long duration of her symptoms, render it probable that a considerable increase of muscular substance had occurred; but these signs are not inconsistent with an extreme degree of dilatation, to which the apparent extent or bulk of the heart was probably in a great measure due, and the effects of which were otherwise so manifest.

This very disproportionate dilatation satisfactorily explains all the phenomena of the case, as well as the total inefficacy of treatment. The hopelessness of such cases is in exact proportion to the dilatation, or enfeebled power of the heart. Depletion can be of service only in so far as it disburdens the embarrassed heart of the excess of blood which it struggles to propel; and the instant it causes any impairment of the power of the already enfeebled heart, the embarrassment of its action, and the consequent obstruction to the circulation, is increased. I am disposed to disapprove of the cuppings practised in this case;—it is rarely necessary to take blood in this way. When it is desired to remove any quantity of blood, a vein should be opened; but in the majority of cases, when the heart's action is embarrassed, four or six leeches will have the effect of allaying the tumult of its action, and give immediate relief.

It is not necessary to multiply cases of this description. Those already narrated will serve to exemplify the direct and powerful influence of dilatation, even when hypertrophy exists; and in a large proportion of cases all the later and fatal effects of diseased heart will be found, as in the preceding cases, to depend upon dilatation.

The rarity of fatal cases in which the hypertrophied heart is as yet little dilated, depends upon the well-known difference between the effects of *hypertrophy* and *dilatation*. The *former* acts temporarily, and causes local effects upon some distant organ, which usually has been weakened by pre-existing disease—*e. g.*, rupture of the arteries or substance of the brain, and consequent apoplexy. The *latter* causes more permanent effects on the entire system, through the impeded circulation.

SECTION II.

ANEURISM OF THE HEART.

Aneurisms of the heart, especially certain forms of the lesion, illustrate very remarkably the influence of dilatation in causing the leading features, and the ultimate evils, of cardiac diseases. The application of the term aneurism is here restricted to a dilatation which is partial, and more or less intimately connected with a degenerated or transformed state of the walls of the heart. I have found

it impossible to compress the cases so much as is desirable; because though, in this paper, only certain points in these cases are discussed, it appears necessary to give an accurate and detailed account of the facts, that they may be available in reference to other questions than those involved in the present inquiry.

I ought to mention that I was prevented being present at the dissections of the two succeeding cases (V. and VI.); but, in both, the hearts were preserved for my inspection.

Case V.—*Aneurism of the left Ventricle, true and sacculated; Dilatation of the Heart; Hypertrophy; Fibrous Transformation of the Muscular Substance; Death by Apnœa.*

Margaret Sheriff, æt. 30, married, was admitted into hospital on the 20th November 1847. She was labouring under most urgent dyspnœa and extreme general dropsy. Her lips were livid; the pulse was small and feeble; the abdomen was swelled and tender; urine scanty, density 1·015, albuminous. She wore a tracheotomy tube.

History.—She stated that troublesome symptoms had arisen within a year; but she could not give a distinct account of their progress. Tracheotomy had been performed many years previously.

In the preceding January she was in hospital under the care of Dr George Paterson, to whom I am indebted for an account of the symptoms and physical signs which then existed.

At that time she suffered chiefly from dyspnœa, cough, and occasional palpitation. Urine was copious, of density 1·015, and free of albumen; pulse was 84, small and weak.

She stated that, with the exception of slight catarrhal symptoms, which she ascribed to the state of the trachea, she had had good health till the preceding November (1846), when, in consequence, as she supposed, of exposure to the weather, aggravation of her chest symptoms, with pain, occurred. Slight swelling of the limbs occasionally existed, and scanty urine. The account of the physical signs, as then observed, will be incorporated with the statement of those noticed by myself in November. She remained only five days in hospital, and was dismissed by her own desire.

From that time she was subject to an often recurring dropsy, which yielded to ordinary diuretics. She was under the care of Dr Thomas Keith, who says that the urine was free of albumen; but he did not examine it within three months of her admission; and about that period the dropsy yielded less readily, and latterly resisted the diuretics.

Physical Signs.—These were ascertained with difficulty, from her distressed and exhausted state, and from the intensity of general bronchitis. They differed from those observed in January, only in the *force* of the heart's impulse, which had become much less; for it was now feeble compared with its extent, and with the extent of the præcordial space dull on percussion. The following was the state of the signs as observed in January, and corroborated in November:—

The præcordial space dull on percussion was extended. The impulse of the heart was diffused, originally of good strength, but it became feeble compared with the extent of the præcordial space. The radial pulse was small. A murmur accompanied the first sound, and was most distinct at the apex. The usual signs of general bronchitis existed; these latterly were intense.

Progress.—Gradual and progressive exhaustion followed, without any material change in the symptoms, and she died on the 26th.

Post-mortem Examination.—Dropsy was general. The larynx, trachea, and lungs, presented no appearance which was unusual under the circumstances.

The heart was enlarged; it had an irregularly globular form, with two cir-

cumscribed tumours, the size of walnuts—one at the apex, the other an inch and a-half to its left. The organ, with a small portion of adhering pericardium, weighed nearly 18 ounces. On laying open the left ventricle in the usual manner, by separating its left wall from the septum, its capacity was found to be greatly increased,—the increase being in a great measure due to a diffused, but partial dilatation or *true* aneurism of the left wall. The endocardium, especially of the dilated left wall, had an opaque pearly appearance.

The tumours of the apex proved to be two well-formed *sacculated* or *false* aneurisms. They communicated freely with the ventricle,—the one in the precise situation of the apex, by a wide orifice; the other to the left, by a narrow circular opening. The cavity of the latter was diminished by a dense coagulum in layers; and the rim of its orifice, and the neighbouring tissues, were rigid and semi-cartilaginous. One lip of its orifice was formed by one of the reticulated muscular bundles which had undergone a partial fibro-cartilaginous transformation. The sac at the apex and the neighbouring parts had a more fibro-membranous structure. Both sacs, as well as the ventricle, contained loose coagula. Their lining was smooth and continuous with the endocardium. Their walls were about a line in thickness.

In the posterior bundle of fleshy columns, and at their base, there was an irregular digital depression or small aneurism; it had a smooth lining, which was continuous with the endocardium. The leaves of the mitral valve were not appreciably altered; the tendinous cords were slightly shorter than usual.

The muscular substance of the heart had undergone extensive transformation of structure. This was most marked around the orifices of the sacculated aneurisms, and in the dilated or aneurismal left wall. In the vicinity of the sacculated or so-called false aneurisms, the transformation was semi-cartilaginous. In the left wall of the ventrical the transformation was fibrous in a very marked degree; the muscular substance was superseded by this dense grey fibrous structure to the extent of nearly three inches; and the average thickness of the wall was not thereby lessened, as it equalled half an inch. The pericardial surface of this portion of the heart was marked by adhesion, and a ragged fibro-cellular patch; the corresponding part of the endocardium was pearly and smooth, almost fibrous in appearance, and the usual reticulated muscular bundles were wanting; they ceased near the base, behind the outer leaf of the mitral valve, by an abrupt transverse line, which formed the upper boundary of the diffuse aneurismal dilatation of the left wall; and this line corresponded precisely with the abrupt line of demarcation which separated the transformed fibrous tissue from the natural muscular substance above. In the substance of this fibrous texture there existed, here and there, the remains of muscular fibre in minute portions, pale and disconnected.

The adhesion of the pericardium was anterior, and to the left of the anterior coronary artery; and it extended only partially to the prominent surfaces of the aneurismal sacs at the apex. To the left of the adherent part, and corresponding to the transformed portion of the left wall of the heart, a patch of ragged fibro-cellular tissue existed on the cardiac surface of the pericardium.

The right ventricle was large and dilated; it extended to the apex of the heart; its walls were decidedly thicker than natural.

Case VI.—*Sacculated Aneurism of the Left Ventricle; Dilatation of the Heart; Hypertrophy; Incompetence of the Mitral Valve; Fibrous Transformation of the Muscular Substance; Pleurisy; Death after Progressive Exhaustion, but unexpected.*

Anne Stewart, æt. 25, a plethoric woman, of dissolute habits, and broken constitution, was admitted on the 14th March 1849. She suffered from extreme dyspnœa, cough, frequent palpitation, and uneasiness, with sense of sinking, referred to the scrobiculus cordis, and epigastrium. Her face was puffy and slightly congested; her lips were livid; the pulse small, 129.

History.—The account she gave of her symptoms could scarcely be depended on. She had been liable to chest complaints, with palpitation on exertion, for two years. Tracheotomy had been performed five years previously; and the tube was worn for two years thereafter.

Physical Signs.—The heart's action was excited and violent; the impulse was more remarkable for its *extent* than its *force*. The præcordial space dull on percussion, was extended; but its precise limits were obscured by mammary development, &c. The sounds of the heart were free of murmur over the upper part of the sternum. At the apex, a murmur existed which was subsequently ascertained, when the action of the heart moderated, to accompany the first sound.

Percussion of the chest elsewhere was resonant, except in the inferior right dorsum, where there was incomplete dulness.

The sounds of respiration were harsh, but feeble in the inferior right dorsum. Some wheezing and submucous rattle prevailed in both sides of the chest.

Progress and Treatment.—Mild laxative medicine was given; leeches were applied to the scrobiculus cordis, and she was confined to bed. The heart's action was immediately moderated; and she felt relieved. For about ten days she improved, slightly but decidedly. The urine, however, was obstinately scanty, of the density of 1·015, and albuminous. No dropsy had as yet appeared. The pulse was always small, but distinct and regular, usually 120 in the minute. Paroxysms of urgent dyspnœa now supervened; cough became troublesome; and she began to manifest a preference for resting on the right side; other signs of pleural effusion on the right side were also developed. She was blistered, with immediate relief; and the paroxysms did not recur for several days.

In the beginning of April, her complaints became rapidly more urgent. Dyspnœa, palpitation, sense of sinking with epigastric pain, occasional sickness and vomiting, occurred. Treatment afforded only temporary and slight relief. Increasing weakness became apparent; the pulse was steadily losing strength; and œdema made its appearance in the lumbar region and feet. From this time she lost ground. She had great general distress; and during the last ten days of life, she had unceasing, restless, wandering delirium, with great exhaustion; and the pulse was irregular. She expired, unexpectedly, on the 17th April, immediately after conversation with the nurse. She preserved a degree of intelligence, when roused and addressed, to the last.

Post-mortem Examination.—Dropsy existed, chiefly in the lower extremities.

Both lungs were partially adherent; and the right pleura contained above half a gallon of turbid serum, with lymph in loose flakes, and adherent membranous layers coating both pleural surfaces. This lung was compressed, not condensed.

The heart was large, and more or less globular; it weighed 14 ounces. Externally, on the posterior surface of the left ventricle, close to the septum, there was a diffused oblong tumour, occupying the two upper thirds of the ventricle, and bounded beneath by a tranverse sulcus, in which there was a small adhesion of the pericardium. The cavity of the left ventricle was dilated; the external tumour depended on a circumscribed aneurism as large as a bantam egg. From its situation, the walls of this aneurism were formed partly by the septum posteriorly, and partly by the left wall of the ventricle. The posterior edge of the mitral valve and its tendinous cords stretched across the orifice or communication of the aneurism with the ventricle; and this communication was large and free. The aneurism contained loose coagula, such as filled the ventricle. The sac projected chiefly at its lower part, and downwards; its walls were uniformly thin,—about a line. Within the sac, on its upper part or roof, there was a patch half-an-inch in diameter, depressed, and forming, as it were, the commencement of a saccule within the aneurism. This patch was coated with a grey friable adherent membrane: its edges were sharp, and discoloured; but loss of substance by ulceration could not be satisfactorily demonstrated.

The leaves of the mitral valve were opaque, not thickened, they were widely

parted; and the tendinous cords were unusually short. The muscular substance of the heart was generally, but slightly and irregularly, hypertrophied. Posteriorly, from the lower boundary of the aneurism to the apex, the muscular substance was incompletely transformed into a fibro-elastic like tissue. In this transformed tissue, there existed several minute lardaceous tumours; and one of the intermuscular sulci was dilated into a commencing aneurism.

The right ventricle was dilated; and extended to the apex of the heart. Its walls were uniformly, but only slightly, thickened. The fleshy columnæ and reticulated muscular bundles were hypertrophied.

The liver was congested, and had the nutmeg appearance.

The kidneys were congested; they were little changed in their bulk; but they were pale in their cortical portion; the pyramids were of a dark red colour, with marked and abrupt line of demarcation from the cortical portion.

Remarks.—The facts of these cases suggest three distinct questions. The diagnosis of cardiac aneurisms is confessedly most uncertain; and, the physical signs in both these cases having been observed with some care, we have to inquire whether any sign or series of signs existed, which may be regarded as distinctive of cardiac aneurisms. Secondly, in regard to the more immediate object of this memoir, these cases indicate the relative influence of true dilatation of the heart, and of the partial dilatation or aneurism in causing the secondary lesions on which the progress and result of the cases depended. In the third place, the lesions of the heart in these cases afford some information regarding the anatomy of this disease, and the mode of origin of cardiac aneurisms.

Before entering upon these questions, it will be well to observe the *forms* which the aneurisms assumed; because the different forms are intimately related to differences in the physical signs, as well as in the secondary pathological effects. Cardiac aneurisms have been distinguished according as they do or do not cause external deformity of the heart; but I cannot see any practical good arising from such a distinction. They have also been distinguished into acute and chronic; and this distinction has, perhaps, a more sound basis; but it involves the question of the inflammatory origin of these aneurisms, which is less directly related to the immediate object of this memoir. Besides, the present series of cases indicates the necessity of distinguishing cardiac aneurisms according to differences which are physical rather than pathological. In the present cases, the aneurisms presented two distinct forms; and they were not more distinct in form than they were in their physical signs, and in their pathological effects. The one is circumscribed, limited, and more or less *sacculated*, with or without a narrow orifice, and was well exemplified by the aneurisms at the apex of the heart in case V. This form has been designated the *false*, or false consecutive, aneurism, both of which names are objectionable, as they involve a theory of which the evidence is not very complete. The other form, as exemplified in case VII., and also in the left wall of case V., is diffused, having no very defined separation from the ventricle, and forming, in

a more correct sense, a partial dilatation of the cavity, or *true aneurism*. In the remarks which follow, I shall observe this distinction, which has an important bearing on the living pathology of the disease. The former is designated the *sacculated* aneurism; the latter the *true* aneurism of the heart. It must not, however, be supposed that cardiac aneurisms always present the one or other of these forms. Case VI. exemplifies the combination of the diffused and circumscribed form in such a way as to justify a third or intermediate class, which may be designated the *mixed* aneurism of the heart. This subdivision is of minor importance; because, evidently, aneurisms of this form will induce results differing from those of the two primary forms only in degree.

In neither of these cases did there exist any physical sign which is not familiarly known to arise in other forms of disease of the heart. In both cases, there existed a murmur with the first sound, which, however, was not to be distinguished from the murmur of mitral incompetence. In the woman Stewart (case VI.), from the appearances described, there can be little doubt the mitral valve was incompetent, and gave rise to the murmur in question. In the woman Sheriff (case V.), the post-mortem appearances did not indicate incompetence of the mitral valve; and the endocardial murmur with the first sound at the apex, must be attributed to the aneurisms in that situation. The only peculiarity noticed in the murmur was its greater distinctness at the base of the heart than is usual with murmurs loudest at the apex. This, however, does occasionally occur in murmurs from mitral incompetence; and a circumstance so trivial and uncertain is really unimportant.

It has been suggested, that under possible circumstances, a double "to and fro" murmur might occur in aneurism of the heart. I am not aware that a double murmur has ever been observed; it at no time existed in these cases; and the form of the sacs, as well as their narrow orifice, appeared fitted to create the double murmur, if its existence were possible:—A double "to and fro" murmur would perhaps assist in distinguishing these aneurisms, while it is impossible to distinguish the single bellows murmur, having its seat in the orifice of an aneurism, from one having a very different source.

The other physical signs in these cases were identical with those which are known to characterise hypertrophy and dilation,—the latter especially is known to preponderate from the feebleness of the cardiac impulse, the horizontal extent of the præcordial space dull on percussion, the softness of the radial pulse, and from the mutual relations of these signs to each other; and these signs must of necessity arise in cases, such as the present, in which the following conditions existed:—An extensive degeneration of the muscular substance of the heart; an unyielding and unwieldy state of the organ, arising out of the rigid and deformed state of its walls; and above all, the increased capacity which must result even from partial dilatation.

These statements are not intended to imply that an endocardial murmur may not arise in connection with a cardiac aneurism, which is neither sacculated nor narrow in its orifice. But it does appear that the murmur arising in the sacculated aneurism is not to be distinguished from that arising from incompetence of the mitral valve; and that the signs of the true aneurism, or partial aneurism, are more or less identical with those of true dilatation. Besides, in both these cases, true dilatation did exist as a complication of the aneurisms; and its signs were of necessity present. Indeed, it is probable that the signs of cardiac diseases in such cases will become decided only when the degree of partial dilatation is great, or when true dilatation is superadded.

In reference to the second question proposed, the secondary effects of the heart-disease in these cases were referable to the dilatation. The sacculated aneurism appears to have been comparatively uninfluential; and the true aneurism, or partial dilatation, acted injuriously only in so far as it approached in degree to the true dilatation, causing lividity, dyspnœa, dropsy, &c., which are well known, and have been shown in the first section of this memoir (cases I. to IV.) to depend on an obstructed circulation from preponderating dilatation.

Let it be borne in mind that, in both these cases, the partial dilatation was such as materially to increase the capacity of the ventricle; and to produce physical effects altogether undistinguishable from those of true dilatation. In both these hearts also more or less general or true dilatation of this and the other cavities had actually taken place. It is reasonable, therefore, to infer that the embarrassed circulation and its usual effects, lividity, dropsy, dyspnœa, &c., which we have seen to arise from a preponderating dilatation, depended upon the same cause in these cases. On the other hand, the circumscribed sacs were such as to cause no material increase of the capacity of the ventricle in one of the cases only (case V.); and the effects of obstructed circulation which were more marked in this patient, depended on the greater amount of the diffused partial dilatation of the ventricle, and on the degree of the true dilatation. We cannot, therefore, attribute to these sacculated aneurisms effects so formidable; which are, moreover, perfectly accounted for by the otherwise dilated heart.

We have observed, that when the effects of dilatation once fairly set in, the progress is uncontrollable, and the case not usually very lasting. In the woman Sheriff (case V.) these effects appear to have arisen above eleven months before the fatal event; and this must be regarded as an unusual length of time for a patient to survive the well-marked effects of dilatation. From the fact, however, that she remained only five days in hospital, when she was first admitted in January, apparently labouring under these effects of dilatation, it may be assumed that the circulation had not as yet become obstructed in a very great degree. It cannot be precisely determined what the

apparent degree of dilatation was at that date; but, from Dr Keith, we have ample evidence that, long subsequently, and indeed within a comparatively short period of death, an unfavourable and rapid change had occurred in the symptoms attributable to the dilatation:—The dropsy increased, and was uninfluenced by treatment; diuretics failed of the effect they previously produced; and the urine became scanty and albuminous.[1] In the woman Stewart (case VI.), the apparently recent origin of urgent symptoms, and the rapid progress of the disease in the later stage, contrasted with the slow and gradual progress of Sheriff's case; and was probably, as in case II., in some measure due to the inflammation of, and effusion into, the right pleura. The conditions directly referable to dilatation of the heart, *e.g.*, dropsy, small pulse, &c., did not exist in the same extreme degree as in case V.; and, *probably*, as observed post-mortem, the dilatation had not attained the same disproportionate excess.[2] The symptoms of pleural effusion became prominent before dropsy, or other extreme effects of embarrassed cardiac action were very evident; and this state of the pleura, as seen in case II., no doubt aggravated the effects of the heart-disease;—prematurely inducing the fatal event.

In the account of such cases by other writers, the structural changes of the heart have been recorded with minuteness, and the anatomy of cardiac aneurisms has received greater attention than any other part of the subject. In these observations, therefore, which it is desirable to make short and practical, I shall confine myself to a short notice of the transformation of the muscular substance, the probable existence of endocardial ulceration, and the pericardial adhesion.

The chief interest connected with the transformed, fibrous state of the muscular substance, is involved in the investigation of its influence in causing the aneurismal state of the heart—true or sacculated. It has been well remarked by Dr Craigie, that it is difficult to say how far this transformed state of the muscular substance is a cause or an effect of the aneurisms. The facts of our cases throw an imperfect light on this question. They do not prove that the sacculated aneurisms were not preceded by a transformation of the tissues of

[1] By this statement, I do not commit myself to any opinion as to the connection between disease of the heart and the albuminuria, which so frequently accompanies its last stage. There is some difficulty in determining how far this state of the urine is connected with a disorganised state of the kidney, which I am disposed to believe usually exists.

[2] This statement is expressed thus cautiously, not so much from any doubt I have in making it, as from the known difficulty in comparing the relative disproportions of hypertrophy and dilatation in different cases. For we know that the embarrassment of the heart's action, arising from disproportionate dilatation, bears no stated relation to the bulk or weight of the organ; and that a smaller heart may be either less or more dilated in proportion than one of greater size.

the heart; but they show that transformation had taken place in that part of the heart where the true aneurism or partial dilatation existed, and that that transformation was antecedent to the formation of the true aneurism, because it existed in parts where dilatation had not as yet occurred; and in regard to the sacculated aneurisms, the saccules which existed in both cases indicate that they may occur without any so peculiar and complete transformation of the tissues as appeared to precede the true aneurisms. If these inferences from the facts of the cases are admitted, it may be stated, in general terms, that the occurrence of true aneurism is determined in a great measure by the transformed state of the tissues; and that the sacculated aneurisms, though they may be preceded by a similar transformation of the tissues, appear to have other causes, and may arise where no adequate transformation exists. These sacculated aneurisms, however, probably tend to induce absorption of the tissues in a greater degree than the true aneurisms do—causing the thin fibro-membranous sac walls. It has been already shown, that the effects of these aneurisms are very much in proportion to the degree in which they cause increased capacity of the heart; now, the small size of many of these sacculated aneurisms, even when very prominent, will probably render their duration so much longer than that of the true aneurisms, as to give time for more advanced changes in the tissues involved in their formation. The fibro-cartilaginous state of the parts in the vicinity of these sacs is a further evidence of this.

Ulceration has been supposed to determine, in some degree, the seat and development of sacculated aneurisms; but there is a want of facts substantiating this; and in neither of the preceding cases was there any good reason to believe that ulceration of the endocardium had existed. Several of the rudimentary saccules were still at a stage of their formation, in which the effects of ulceration would have been apparent if it had ever existed. Their surface, however, was continuous with the endocardium; and in some of them the margins of the orifice were formed by muscular bundles, little altered, and having no appearance of ulceration. In the interior of the aneurismal sac, in the case Stewart (VI.), there was a patch which, from the sloughy state of its surface, and from its sharp, abrupt edges, with discoloration, almost justified the opinion that it was an ulcer, but this was doubtful. The loss of substance was not apparent; but this portion of the sac had an additional depression, as it were, of a commencing dilatation. The only inference which these facts admit of is, that the sacculated aneurism occurs without antecedent ulceration.

Pericardial adhesion existed in both these cases. It was very limited in the case Stewart (VI.); and it was seated at the posterior surface of the heart. In the case Sheriff (V.) it existed anteriorly, and was extensive.

I am disposed to think that undue importance has been attached to adhesion of the pericardium, from the supposition that it tends

o prevent rupture of the sac and hemorrhage. It is said to have existed chiefly in cases in which the sacs formed prominent tumours; nd that it is due to the pressure of the opposed pericardial surfaces y the tumour. There is an insufficiency in this explanation; and he cases before us not only cast doubt upon it, but appear to afford ata for a more rational notion of the nature and relations of this morid state.

Observe, in the first instance, that the adhesion was neither limited the surface of the tumours, nor did it involve these in any remarkble degree. Secondly, The part of the pericardium which was most dherent, especially in case V., or had the roughened surface of a bro-cellular formation, corresponded precisely with that part of the uscular substance which had undergone the fibrous transformation; nd the opacity of the corresponding part of the endocardium, as well s the destruction of the reticulated fleshy bundles, showed that the ltered action in which the transformation of the muscular tissue riginated, had extended to and involved the whole thickness of the eart's walls, and resulted in the adhesion of the opposed pericardial urfaces.

It cannot be denied, that adhesion might possibly arise from pres-ure of the aneurismal tumour. In both these cases, however, we see nsiderable tumours without this effect, and the adhesions of the sacs hich did exist were contiguous to that which corresponded to the ansformed tissues, and involved neither the most *prominent* nor the *innest* part of the sacs.

In the case Stewart (VI.) there was a marked disproportion be-ween the extent of the transformation of the muscular substance and e limited adhesion. The surface of the sac was devoid of all adsion; and the transformation of the tissue, though extensive, was t by any means complete, and had not attained nearly the degree hich existed in case V. This, perhaps, explains the as yet unafcted state of the pericardium; certainly, where it did adhere in the lcus beneath the sac, the transformation of tissue was more comete than elsewhere.

SE VII.—*Diffused or True Aneurism of the left Ventricle; Dilatation of the Heart; Hypertrophy; Fibrous Transformation of the Muscular Substance; Hydrothorax; Chronic Pneumonia; Death by Syncope.*

Mrs Richards, ætat. 66, a delicate-looking woman, was admitted on the 5th ne 1847.

Dyspnœa was not urgent, but she was unable to rest continuously in the rizontal posture; and she had cough and palpitation. She died suddenly a w hours after admission, before any careful examination of the case had been ade.

History.—This could not be ascertained, farther than that she had been liable palpitation for a period of years.

Post-mortem Examination.—The volume of flesh was good. There was geral dropsy, and the right pleura contained a gallon of serum. The lower be of the left lung was condensed in the second stage of pneumonia.

The heart was rounded in its external form, chiefly from a diffused bulging of the left wall; the cavities were filled with loose dark-coloured coagula. The weight of the organ was thirteen ounces, and it was dilated in all its cavities. The capacity of the left ventricle was more especially increased by the diffused aneurismal dilatation of the left wall, the muscular substance of which was almost completely transformed into fibrous tissue, presenting a mere trace of muscular fibre. This wall of the left ventricle was, in consequence, rigid and fibro-membranous to the extent of three inches; and the ventricle did not collapse when laid open. The dilated fibro-membranous wall was little more than one line in thickness.

Remarks.—Great variety has been observed in the age of individuals affected with cardiac aneurisms; and it is probable that the disease will be found to assume different forms at different periods of life. In this case the atrophy of the dilated part, and the removal of the muscular substance, was, perhaps, more remarkable than the transformation of the tissues, though it is possible that transformation may have preceded the atrophy of the left wall. Still the existing state of the heart contrasts remarkably with the fleshy fibrous transformation in the two preceding cases, both of whom were in comparatively early life.

The mode of death in this case, by syncope, is not unusual in cases where the heart is dilated; and there can be no doubt that the capacity of the left ventricle, and its embarrassed action, had much to do with this accident, though, as in case II., the state of the lungs and pleura was probably not without its influence.

The next case was not under my own observation, having occurred in the practice of Mr Benjamin Bell, who kindly favoured me with the details, from which the following account has been drawn up.

CASE VIII.—*True Aneurism of the Left Ventricle of the Heart: Dilatation; Hypertrophy; Fibrous Transformation of the Muscular Substance; Pulmonary Apoplexy; Death unexpected.*[1]

Mr ——, æt. 65, a professional gentleman, of full body, but without plethora, temperate, and of active habits, had a severe attack of acute pneumonia in the right lung in December 1844 (four and a-half years before death), and he was re-attacked by the same disease in the following summer, 1845. He was largely bled for both attacks. The lung appeared to recover its healthy action; but subsequently, more or less breathlessness on ascending even a slight acclivity existed; and, though his mind continued active as ever, he found himself unfit for the exercise he had been accustomed to. This may be regarded as the beginning of Mr ——'s ill health.

Subsequently, the urine occasionally became scanty; but this was readily counteracted by ordinary diuretic remedies. He was subject to uneasy feel-

[1] This expression requires explanation. It is intended to convey the idea of death anticipated as perhaps not very distant, but nevertheless occurring at an unlooked-for moment. This unexpected death is distinguished from sudden death, inasmuch as in the latter a fatal issue was less immediately anticipated; and it is equally distinct from death after gradual exhaustion.

ings in the chest—sometimes of the right side, sometimes in the cardiac region, which, however, subsided after leeching, a small blister, or a belladonna plaster.

About four months previous to death, the breathlessness, and, as he himself called it, "the disability on locomotion," became so much increased, that he was unable to walk a few yards, even on level ground, without stopping to take breath. He referred the painful or disabling sensation to a point near the situation of the gall-bladder.

The chest had been repeatedly examined in the course of the preceding four years; but no sign indicating a morbid state of the heart was observed until latterly, when the impulse of the heart became forcible and extended, and an indistinct murmur with the first sound was occasionally audible at the seat of the apex, and below the left costal margin.

About six weeks before death, the lower extremities became œdematous; urine was at the same time scanty, and diuretics exerted little or no influence. For one or two days the urine was observed to be albuminous; but this afterwards disappeared. About ten days previous to death the anasarca suddenly subsided, without either purging or diuresis; and then hæmoptysis occurred, which never left him. At this time the dyspnœa was much aggravated. To the last he was conscious, and to a certain degree intelligent when roused; but he readily lapsed into a state of dull, wandering lethargy. Shortly before death he was in his usual state, extremely feeble and exhausted; but at the last his death was instantaneous, and unexpected by his family.

Post-mortem Examination, thirty hours after death.—The surface generally was slightly yellow. The volume of flesh was considerable; with more than the usual amount of fat under the integuments.

The heart was large and globular, weighed eighteen ounces, and was distended in all its cavities by loose dark-coloured clots. In the apex of the left ventricle, there was found a fibrinous coagulum, firm, grey, and adherent; it was contained in a partial dilatation of the ventricle, which caused a pouch-like addition to the cavity. The ventricle was immensely increased in its general capacity, partly by this sac, and partly by general or true dilatation. The widest diameter of the sac was at its orifice; its lining was continuous with the endocardium, and had one or two patches of yellow atheromatous deposit; the reticulated muscular bundles were mostly wanting in its interior. The wall of the sac was formed in part by the septum of the ventricles, and by the left wall, at the apex; it was attenuated and fibro-membranous, without a trace of muscular tissue. A small but firm adhesion of the pericardium existed; surrounded by a fleshy cellular patch, on the surface of the tumour, where the wall of the sac was thinnest. The walls of the ventricle elsewhere were thickened; in the anterior part, their muscular substance had undergone a very imperfect transformation into fibrous tissue; and the fleshy columns of the ventricle were similarly, but more completely, transformed. The endocardium, especially towards the apex, was opaque and thickened. The valves were normal, except the aortic, which, as well as the aorta and coronary arteries, were altered by a calcareous and atheromatous deposit. The right ventricle was dilated; the reticulated muscles hypertrophied, and the walls slightly thickened. The auricles were large.

The lungs were partially emphysematous anteriorly. In all the lobes there were masses of condensation from hemorrhagic effusion—some of considerable size.

The liver was small and pale. The kidneys normal.

Remarks.—The name and known accuracy of Mr Benjamin Bell is a guarantee for the correctness of the narrative of this case; and the length of time that the patient was under observation, gives a value to the *history* which cannot be claimed for cases observed only during the later stages. I shall, therefore, endeavour to indicate the

relation between the different forms of heart-disease which existed, and the morbid phenomena noticed at different stages.

The case illustrates, very strikingly, the influence of dilatation; for this was the only lesion of the heart competent to explain the symptoms; and the dilatation was of two kinds—it was originally partial or *aneurismal;* latterly, *true* or general dilatation also existed. It appears probable that the slight degree, and gradual progress, of the symptoms in the first years, was due to the *former;* while the extreme obstruction to the circulation, and the rapid progress of the symptoms during the last few months, were due to the *latter*. The state of the parts proves that the aneurism was of old standing; that it must of itself have increased the capacity of the ventricle, and must otherwise have embarrassed the action of the heart;—causing effects similar to those of true dilatation, but less in degree, just because the dilatation was less or partial. The fact that the lesion involved the effects of dilatation from so early a date, is, I believe, intimately connected with the steadily unfavourable progress of the case.

The proof that the true dilatation was of more recent origin, is less direct. A very decided aggravation of the symptoms occurred within the last six months; and the only lesion accounting for this, was the great dilatation of the left ventricle. This increased capacity was not due to the aneurism of the apex alone, but also to true dilatation of the whole ventricle; and the aneurism presented no appearance of having undergone any change or advance such as would explain the recent aggravation of the symptoms. While, therefore, the old standing aneurism was the apparent and sufficient cause of the pre-existing slighter symptoms, the true dilatation was probably connected with the development of the later symptoms of a more obstructed circulation; though the date of its origin cannot possibly be determined, nor its precise influence on the progress and issue of the case distinguished from that of the aneurism. Nevertheless, we see secondary pathological effects and symptoms in the case which an uncomplicated partial dilatation scarcely explains, and which are identical with those we have seen to accompany simple preponderating dilatation (cases I. and II.). The dilatation of the ventricles, especially the left, was the leading feature of this case; and, as in other cases, it co-existed with an instantaneous and more or less unexpected death, a distended state of the heart by recent loose clots, and pulmonary hemorrhage.

The hypertrophy of the right ventricle was, perhaps, a cause of the slow progress of this case in the early stages. But this question will be considered in commenting upon other cases.

The wall of the aneurismal sac, as in case VII., was attenuated and membranous, but to a less extent. This fact is worthy of remark in connection with the age of the patients, but is quite insufficient to establish any necessary relation between this state and advanced life. Moreover, the muscular substance of the heart was

elsewhere in progress of the same fibrous transformation, as in cases V. and VI.

The state of the pericardium throws some light on the probable cause and mode of the adhesions in these aneurisms.

The tumour caused by the aneurism, on the external surface of the heart, had no very manifest prominence distinct from the globular and dilated state of the organ generally; the adhesion of the pericardium existed at the apex, but no such protuberance of the tumour existed here as to justify the idea that increased pressure had existed; it is probable, therefore, that some other cause than pressure had to do with the formation of this adhesion. Besides, the band of adhesion was in itself small and insignificant; but it was surrounded, on the surface of the tumour, by a considerable cellular fleshy patch; and the sac wall was most attenuated at, and near to, this situation. It appears, then, first, that the thinness of the sac wall, just as is observed in other hollow viscera, had to do with the exudation on the pericardial surface; and, second, that the exudation may result merely in the formation of a layer or patch externally; while adhesion depends upon the accidental circumstances of contiguity, &c. It is manifest, that the formation of an organised layer externally, by thickening the attenuated walls, will have the effect of delaying rupture of the viscus; and this hindrance will be promoted by the adhesion of the opposed surface, should that *accidentally* occur.

The entire failure of the diuretics, in the late stages, is an almost invariable occurrence, and was well seen in the progress of case V. It is probably intimately connected with, or an effect of, dilatation.

The sudden disappearance of dropsy shortly before death is not infrequently observed. The practical lesson which the circumstance teaches is, that other secondary morbid conditions, such as the hemorrhagic condensation of the lung in the present case, having been established, the removal of dropsy, viewed singly, does not materially modify the prognosis.

SECTION III.

CONTRACTIONS OF THE VALVES.

It will not be difficult to show, that the symptoms which mark the progress of cases of valvular disease of the heart, arise less directly from the valvular lesions than from the hypertrophy and dilatation which so invariably supervene. Further, it appears that dilatation is the more immediate cause of the secondary evils which usually arise in these, just as in other cases; and it is probable that hypertrophy has, in the first instance, a counteracting influence on the effects that

might arise from the deformed or incompetent valves; but frequently it is attended by those untoward circumstances which have been termed accidental, and with which the valvular lesion is indirectly connected.

Case IX.—*Contraction of the left Auriculo-Ventricular Orifice; Dilatation of the Heart; Hypertrophy; Death by Apnœa.*

Mrs Forrester, æt. 38, a married woman, of spare development, was admitted, February 10, 1848, with urgent dyspnœa, respirations 36, lips livid, pain, and acute tenderness of the epigastrium; she had frequent paroxysms of violent palpitations, with vertigo; no cough. The pulse was small, hurried, and irregular; urine scanty, and slightly albuminous, sp. gr. 1·023.

History.—She stated that her health had been impaired only for six months, and that she never had rheumatism, cough, nor hæmoptysis. For three months slight œdema had occasionally existed; palpitation and dyspnœa had been only at times so urgent as to prevent her resting in the horizontal posture.

Physical Signs.—The action of the heart was tumultuous. The impulse was double—one diffused and more forcible than the other, which was sharp, quick, and slight. The radial pulse was very small and feeble, contrasting with the extent and force of the heart-beat, with which it was not synchronous. The præcordial space, dull on percussion, extended in the vertical direction from the third left cartilage to the costal margin, and horizontally five inches. The sounds of the heart were, at first, and indeed pretty uniformly, hurried, irregular, and confused, with an undefined prolongation and harshness affecting, though uncertainly, the first sound. Subsequently, when the action of the heart moderated, there was audible, in addition to the prolongation, a soft bellows-murmur with the first sound at the apex. The veins of the neck were neither distended nor pulsating.

The hepatic dulness on percussion extended to the epigastrium and lateral region of the belly.

Progress and Treatment.—She was immediately relieved, the tumult of the heart's action was lessened, and she could rest in the horizontal posture, after leeching in the scrobiculus cordis. Dyspnœa, however, did not entirely cease, and shortly afterwards slight œdema was observed; urine also continued scanty. Diuretics acted very imperfectly and without effect on the dropsy, which already showed a tendency to a steady increase.

In the beginning of March, about five weeks before death, cough occurred, and harsh respiratory murmur, with submucous rattle in the inferior dorsal regions. The paroxysms of palpitation became more frequent, with threatening fits of syncope; the heart's action, always more or less tumultuous, was now perceptibly less forcible; and she was unable to leave bed, though she still had occasional intervals of slight comparative comfort. Dropsy soon became general, and her distress from dyspnœa, and the sense of impending death, more unceasing. During the last ten days dull percussion was well marked in both lower dorsal regions; bowel complaint, sickness, and vomiting, and attacks of approaching syncope, were frequent; and she died exhausted on the 3rd April, after great and protracted pectoral anguish.

Post-mortem Examination.—There was general dropsy of the cellular tissue and cavities. The heart was increased *greatly* in bulk; it was globular, and distended in all its cavities; and weighed 15 oz. The left auriculo-ventricular communication was contracted, admitting the forefinger as far as the second joint; both leaves of the mitral valve were thickened and rigid. All the cavities were dilated—especially the ventricles, and the right most of all. The walls of the left ventricle were above the normal thickness; those of the right ventricle were thin, in some parts almost transparent, and membranous. The left auricle contained a dense fibrous polypus. The muscular substance

of the heart was soft. The inferior lobes of both the lungs were condensed by hemorrhagic effusion. The abdominal organs were not appreciably altered, except the liver, which was slightly puckered on its surface. The kidneys were pale; the capsules slightly adherent.

Remarks.—The lesions of the heart in the preceding case may be reduced to four:—Hypertrophy, and dilatation; contracted, and incompetent mitral valve. The two former constitute one compound lesion, rather than distinct morbid conditions; and in this, as in less complicated cases (Cases I. and V.), the obstructed circulation was due to the preponderating dilatation, rather than to the hypertrophy. We have, however, to inquire, what influence the diseased state of the mitral valve had upon the progress of the case.

Our patient presented the usual effects of obstructed circulation in an extreme degree; and we have seen in other cases all these effects arising from a preponderating dilatation. If it be argued that the contracted orifice would obstruct the circulation, and that it was the cause of that state in our patient, it must be admitted that so very great obstruction of the circulation as existed would require a correspondingly great contraction, but the orifice had a circumference of two inches; and cases exist in which the aperture was more contracted, without any such manifested obstruction of the circulation. It is a reasonable inference, therefore, that, in this case, the symptoms of obstructed circulation were due to the dilatation rather than to the narrowing of the mitral orifice.

The dilated right ventricle has been regarded as characteristic of these cases, and as arising directly and necessarily from the contracted left auriculo-ventricular communication. I must demur to this opinion, partly on the ground of this case, in which we find a *less* degree of mitral contraction complicated by the *highest* degree of dilatation of the right ventricle, and partly on the ground of cases to be hereafter recorded, in which hypertrophy of the right ventricle existed. It appears probable that the state of the right ventricle has an important influence on the symptoms and progress of these cases, and that other causes besides the contracted aperture are concerned in the development of hypertrophy or dilatation. These questions will be resumed, in commenting on other cases.

In many respects the features of this case were similar to those we have already seen in cases of dilatation. The steady and progressively unfavourable advance of the symptoms, and the great amount of the pulmonary apoplexy, were especially due to the dilatation, just as in Cases I. and IV.; and in both these respects this case contrasted with others, to be recorded, in which hypertrophy appeared to preponderate. The attacks of syncope, and the tumult of the heart's action, also depended upon the preponderating dilatation.

While, then, it cannot be denied that these effects may have been in some degree due to the contracted aperture, nevertheless it does appear that they only arose and progressed along with the dilatation

of the organ, which may be assumed, from the signs and from the anatomical condition, to have had a comparatively recent origin and progress.

Tenderness of the epigastrium is more frequently present in this than in other forms of diseased heart; it is ordinarily associated with the signs of increased size of the liver, and sometimes with obstinate sickness and vomiting; and the benefit of leeching in the scrobiculus cordis is always very marked. The enlargement of the liver is for the most part temporary—probably depending on congestion; it does not usually exist after death.

The physical signs, in cases of mitral contraction, are subject to great variation; these cases exemplify, very strikingly, the general truth that particular physical conditions are indicated not so much by individual signs, as by the association of several signs; for it not unfrequently happens that the most usual and characteristic sign is wanting.

In our patient Forrester, the physical indications resolved themselves into those of the valvular disease, and those of the dilatation and hypertrophy. The signs of dilatation were in some degree obscured by those of the hypertrophy and valvular disease, but they were every day more pronounced in the progress of the case. The extent of the præcordial space dull on percussion, especially horizontally, and the progressively lessening force of the impulse, taken in connection with the general symptoms of the case, and the known tendency, in such cases, to dilatation, justified the opinion that dilatation was considerable. The state of the impulse, which is one of the most important signs of dilatation, was less available in this case, in the first instance, owing to the disproportion between the cardiac and radial pulse—the latter being unduly diminished by the mitral contraction; but the subsequent decline of its force afforded the desired information. The signs do not enable us to distinguish very precisely the cases in which the right ventricle is so largely dilated; the extension of dull percussion to the right side of the sternum affords some information, but this occurs also when hypertrophy of that ventricle exists. This difficulty is, I believe, of less importance, as dilatation of the right ventricle, is, so far as I have observed, usually accompanied by a similar state of the left ventricle; and this lesion is readily recognised from its greater influence on the heart's action and the altered state of the systemic circulation.

The state of the pulse in this case was quite distinctive of the mitral contraction. The most important of the signs of the pulse consisted in the *disproportion* between the *force* of the heart's impulse, and the *smallness* of the radial pulse. This disproportion exists in every case of contracted orifice; and it is appreciable, even in cases, such as the present, in which the force of the heart's impulse was diminished by dilatation. It acquires additional value as a sign of this form of disease from the fact, that it is always permanent, continuing even when the patient is in his best state

The irregularity of the pulse and its want of synchronism with the impulse of the heart, which existed in Forrester's case, is frequently wanting; and it was, I believe, due, in her case, in a great degree, to the dilatation, and in some degree to the mitral incompetence.

It was difficult to appreciate the state of the heart's sounds; and it was still more difficult to report or state in writing their actual condition. This difficulty was owing in part to the tumultuous state of the heart's action, and in part to an undefined vagueness of the sounds themselves. When the action of the heart moderated, the murmur of an incompetent mitral valve was appreciable, and the harsh grating state of the first sound, though it was vague, uncertain, and difficult to appreciate, was very important, because there are many cases in which the murmur indicative of the mitral contraction is imperfectly formed, and amounts merely to this vague harsh prolongation, which owes its diagnostic significance to the facts, that it is audible in the lower part of the cardiac space, that it accompanies the commencement of the first heart-sound, or rather precedes it, and appears at first, as it were, to accompany the second sound, and that it has a *harsh* grating, and not the *soft* bellows, tone. This undefined grating state of the sounds, in connection with the disproportionate smallness of the arterial pulse, is conclusively indicative of the mitral contraction, and is not met with in other forms of disease of the heart.

It has been already stated that the contracted valves are also usually incompetent; it therefore happens, in these cases, as in our patient Forrester, that a bellows murmur, with the impulse, accompanies the confused or grating state of the sounds arising from the contraction. This is probably the cause of the erroneous description of the sounds, in contracted apertures, given even in standard works; and it certainly increases the difficulty of analysing the sounds in practice. There are two distinctions between the murmurs accompanying these two forms of disease;—that of regurgitation, or incompetence of the valves, accompanies the impulse and termination of the first sound,—that of contraction precedes, or accompanies, the commencement of the first sound;—the murmur of regurgitation is almost without exception of a *soft* bellows character—that of contraction, is as invariably *harsh* and grating. In practice, there are difficulties in recognising these distinctions, owing, on the one hand, to the altered state of the heart's action and the deranged rythm, and, on the other hand, to the very varying degrees of the *harshness* or *softness* of these murmurs. There is no difficulty in explaining the rationale of these morbid sounds—their rythm and character, and the causes of these—the sole difficulty is in their practical recognition; this is to be done only by the accustomed ear, and much aid arises from the co-existing signs.

I have very frequently found, in cases of largely dilated right ventricles, that the veins of the neck were permanently gorged, and, still more frequently, that they pulsated irregularly. This is, in

some measure, due to the widening of the auriculo-ventricular communication which so invariably occurs in dilatation of the right cavities of the heart. The unaffected state of the veins in Forrester's case is therefore the more worthy of notice; for, in her case, the dilatation of the right side of the heart was very decided; and it may, perhaps, be explained by the thin and feeble state of the muscular substance of the right ventricle.

Case X.—*Contraction of the Left Auriculo-Ventricular Orifice; Hypertrophy of the Right Ventricle of the Heart; Death by Coma.*

Mary Fyffe, æt. 18, was admitted on the 30th of January 1849. She was a well-developed girl, unmarried, and employed as a mill-worker. Her lips were livid; she had slight dyspnœa; and there was œdema of the face and feet. She suffered from epigastric pain and tenderness; and she had trifling cough, without expectoration.

History.—She stated that, for two years, she had been liable to attacks of dyspnœa and palpitation, with general uneasiness, which unfitted her for work. In the intervals of these attacks she had enjoyed comparative comfort; hæmoptysis had never occurred; and she had never had rheumatism.

Physical Signs.—The radial pulse was small and irregular, and contrasted with the extent and force of the heart's impulse. The impulse of the heart extended to the entire præcordial space; and it was not proportionately forcible; —the impulse and situation of the apex was undefined. The space of præcordial dull percussion measured five inches in the horizontal, as well as the vertical diameter; and it extended an inch to the right of the mesial line. The sounds of the heart were confused. The impulse was accompanied by a bellows murmur which was less distinct upwards, over the region of the ventricles; the first sound was also prolonged, harsh, and grating.

The sounds of respiration were harsh, with some sibilant wheezing.

The hepatic dulness on percussion extended a hand's breadth into the abdomen.

Progress and Treatment.—She was leeched, and the bowels were freely acted upon, with immediate relief. The urine was obstinately scanty—10 oz., and faintly albuminous, with a density of 1·022.

On the eighth day after admission, in the morning, she complained of slightly indistinct vision, and of dyspnœa; twelve leeches were applied to the region of the heart; but she rapidly lapsed into a state of coma, which, *at noon*, was complete. The face was congested, and her lips livid; breathing was occasionally and slightly stertorous—10 in the minute, and at times interrupted for several seconds; the pulse was imperceptible. The heart's action was more forcible than previously, and regular. She was bled to 15 oz. from the external jugular vein; and, in the course of an hour, the stupor had, in a great degree, passed off; but, in the course of the afternoon, fatal coma suddenly supervened.

Post-Mortem Examination.—The volume of flesh was normal. There was slight general dropsy.

Head.—The subarachnoid veins were full, not distended; the lateral ventricles of the brain were nearly empty; and the consistence of the cerebral substance was normal.

Chest.—The heart was increased in bulk, and weighed 15 oz. The relative size of the ventricles was reversed; the right, being the chief seat of hypertrophy, formed the rounded apex of the heart. On being laid open, this ventricle did not collapse; the walls were four to six lines in thickness; and the muscular bundles within were unusually large and distinct. The capacity of the ventricle was slightly increased; and it was distended by a loose coagulum. On the leaves of the tricuspid valve, there were numerous minute fleshy ex-

crescences. The left ventricle formed, as it were, a mere appendage to the right; its cavity was slightly larger than natural; its walls were scarcely thickened. The left auriculo-ventricular communication was contracted, so that it admitted the point of the little finger with difficulty; the margins of the orifice were ragged and calcareous. Both auricles were dilated and distended by coagula. The pulmonary artery presented several atheromatous patches.

The lungs were slightly adherent, otherwise normal.

Belly.—The spleen weighed 12 oz., and was firm and fleshy.

The kidneys were congested. The cortical portion was pale, not manifestly granular; the tubular pyramids had the usual dark colour, and were bounded at their base by an abrupt margin.

Remarks.—The leading facts in this case were the manner of death, and the hypertrophy of the right ventricle; in both of these particulars, as well as in the state of the left auriculo-ventricular communication, which was contracted in the highest degree, the case contrasts with the preceding one. In cases of contracted orifices, as in other forms of diseased heart, death occurs in various ways, by apnœa, with progressive exhaustion; or suddenly, with coma, apoplexy, or syncope. Each of these modes of death has reference to other circumstances than the mere state of a particular orifice or valve, and depends to a great extent upon the condition of the muscular substance of the heart.

The powerful influence which the state of the right ventricle exerts on the progress of cases of diseased heart, is indicated, but indirectly, by such cases as case IX. (page 24), in which the dilatation of the left ventricle makes it difficult to determine how far the obstruction of the circulation was due to the dilatation of the right ventricle; or whether the dilatation of the left ventricle may not have been more directly influential in causing it. The present is the only case I have met with, in which so great hypertrophy of the right ventricle existed, without hypertrophy of the left to any great degree; and though we cannot attempt to prove, from a solitary case, that the coma was due to this lesion, still the case, as well as others more or less similar, in which the organic lesions usually accompanying the apoplectic state were wanting, goes to indicate that extreme hypertrophy of the right ventricle may induce suddenly fatal coma. At all events, the contrast in these two cases (IX. and X.) deserves remark; in the former, we have the conjunction of dilatation, especially of the right ventricle, and permanently obstructed circulation; while, in the latter, we see hypertrophy involving chiefly the right ventricle, associated with sudden fatal coma.

There is a well-known class of cases, to which the present one appears to belong, in which the coma is unexplained; it occurs in the course of some cases of renal disease associated with hypertrophy of the heart, and without any such state of urine as might explain it; and I have met with temporary amaurosis in the like circumstances. The invariable existence of hypertrophy in these cases indicates a connection between this state of the heart and the coma, though it be unexplained. In the case of our patient

Fyffe, I do not see any explanation of the coma, except the hypertrophy of the right ventricle; and other cases present a corroboration of this apparent connection. The effect of the blood-letting in this case proves its suitableness; and, in the cases of renal disease above referred to, I have met with even more decided benefit from bleeding. The blood-letting must ordinarily be pushed with greater freedom than the generally feeble state of the patient, or the diseased state of the heart itself, might appear, at first sight, to admit of.

Apart from the unfavourable, and, as it may be termed, the accidental effects of hypertrophy of the right ventricle already referred to, the present case, and the contrast of its progress with that of the one (IX.) preceding, indicates that, while the dilatation of the one aggravated or developed the evils arising from the valvular disease, the hypertrophy in this case had the effect of counterbalancing the obstructive effects of the contracted orifice. The facts on which this statement is founded, are, the greater length of time during which the patient endured, and frequently recovered from paroxysms of cardiac distress, and the completeness of the respite she enjoyed during these intervals; while, in case IX., the symptoms once fairly developed, the progress was almost uninterrupted and unfavourable. Again, but for the counterbalancing influence of the hypertrophy in this case, the obstruction of the circulation ought to have been greater than in case IX. (Forrester), because the supposed obstructing cause, the contraction of the mitral orifice, was very much greater. Accordingly, we see a very different anatomical state of the lungs in these two cases. Pulmonary apoplexy has been supposed to be intimately connected with contraction of the mitral orifice, like obstructed circulation generally; but we find, in the case in which the contraction was greatest, neither hemorrhagic condensation of the lung, post-mortem, nor, according to the statement of the patient, had she ever had hæmoptysis; while, in case IX., both conditions existed—the dilatation being greater, though the valvular obstruction must have been less.

The heart in Fyffe's case was virtually free of dilatation; for, though the right ventricle was increased in capacity, the hypertrophy was much greater in proportion. The effect of this hypertrophy of the right side was to give the heart nearly the same globular form as occurs in dilatation, but its general bulk was less; and the tricuspid valve and pulmonary artery were in a morbid state rarely met with in the right side of the heart.

Such cases as the present are apt to create confusion, in reference to the signs of hypertrophy and dilatation. Our notions of the physical signs of hypertrophy are gathered from a very different class of cases, in which the left ventricle is chiefly involved. In such cases the effects of the hypertrophy and the physical signs are more manifest, because of the greater amount and degree of the hypertrophy, its more direct influence on the systemic circulation, and, its situation being more anterior, when the left ventricle is affected, the car-

diac impulse is more directly influenced. Hence, from the physical signs alone, the hypertrophy was very imperfectly indicated in this case. Indeed, so far as these signs go, they bear a close resemblance to those of dilatation. The horizontal extent of the space dull on percussion; the dulness on the right of the sternum; and the state of the heart's impulse, which was not forcible in proportion to the extent of the præcordial space:—these signs were not to be distinguished from those observed in cases of preponderating dilatation. It, therefore, becomes necessary to take into account the general condition of the patient; and in our case we saw that the circulation had not suffered any such obstruction as would have existed had the above-mentioned physical signs depended on dilatation.

The anormal state of the pulse and heart's sounds depended, just as in case IX., on the contracted and incompetent condition of the mitral orifice.

Case XI.—*Contraction of the Left Auriculo-Ventricular Orifice; Hypertrophy of the Heart; Dilatation; Chronic Bronchitis; Pleurisy; Death unexpected.*

John Glendinning, æt. 22, was repeatedly under observation from March 1845 till the time of his death, 23d November 1848. He was a young man of slender frame, but well developed, except in the chest, which had an irregular prominence in front, and he had permanent venous injection of the face.

He stated that cardiac symptoms had existed for about a year previously, so that the progress of the case extended over a period of five years. He was originally employed as a gardener; during the latter years of his illness he attended to a turnpike gate, on a road not much frequented.

He was placed under my care, in March 1845, by Dr Richard Mackenzie, with the usual symptoms of obstructed circulation—viz., intense dyspnœa, œdema of the feet and ankles, distention of the belly, &c.; and he was subsequently liable to similar attacks, though they were not usually so severe nor so enduring as this one. The leading symptoms of these attacks were *dyspnœa*, with congestion of the face, great substernal distress, palpitation, vertigo, and the sense of impending suffocation; acute *epigastric tenderness*, with extended hepatic dulness on percussion, tympanitic fulness of the belly, and vomiting; *œdema* of the feet and ankles; *scanty urine;* and *cough*, which was not severe until latterly. These attacks occurred for the most part when the weather was cold and changeable; they lasted at times only a few days, sometimes two or even three weeks.

He always derived benefit from rest; leeching in the scrobiculus cordis invariably relieved the distress and pain of the upper part of the belly; laxatives, expectorants, and diuretics, were also used with various effect; diuresis was induced very uncertainly, but always relieved him.

The intervals of the attacks were of very various duration; a second attack rarely occurred within two or three months; and during 1847 he had very long-continued respite. Latterly, even when in his best state, a slightly-laboured breathing existed permanently.

Physical Signs.—The præcordial region was unusually prominent. The impulse of the heart was visibly extended and forcible; the apex beat in the fifth intercostal space, four inches to the left of the mesial line. The veins of the throat were distended and pulsated. The præcordial space dull on percussion was four inches square, and extended to the right side of the sternum. At the seat of the apex, a strong vibration, or fremitus (purring tremor), was communicated to the hand, not corresponding precisely with the impulse. The sounds of the

heart were confused and undefined, especially over the ventricles. At the apex they were superseded by a double murmur—the one harsh, grating, and vibrating; the other, soft and blowing, distinctly accompanied the impulse of the heart. At the base of the heart; the first sound was faint, the second was perfectly pure. The radial pulse was irregular, not synchronous with the heart-beat, and small—contrasting with the extended forcible beat of the heart.

During 1847 and 1848 (the last two years) the cardiac signs underwent great change. The harsh grating murmur ceased; and the impulse of the heart fell to forty in the minute; it was forcible, corresponding to the radial pulse, and each beat was accompanied by a soft bellows murmur; in the interval of these beats, a quick tumbling action was *audible*, scarcely perceptible to the hand, and not causing any arterial pulse. Latterly, the irregularity became less intermittent, the tumbling action ceased; the impulse had a more uniform force, but was still very irregular in frequency, as well as in size—about 90 or 100 in the minute.

Termination.—His death was unexpected. He had been well for some months, and on the 22d November 1848, he sent for Dr Buchanan, who found him in great distress, with the usual symptoms of his attacks; he had been unable to lie down for two nights. The usual treatment was commenced; but he sank unexpectedly during the morning of the 23d. No precise account of the symptoms at the last could be procured from the attendants.

Post-mortem Examination.—The volume of flesh was rather small; the face was livid; there was no dropsical swelling.

Both pleuræ were extensively adherent; some recent soft lymph and seropurulent fluid existed on the right side. Both lungs were voluminous and emphysematous—the right more so; they crepitated everywhere, but were slightly gorged with blood in the lower lobes.

The heart, while still in the body, appeared of great bulk, owing to the distended state of the auricles, especially the right; it had an oblong globular form, and its rounded apex was formed by the right ventricle, which was much hypertrophied; the left ventricle also was hypertrophied, but less in proportion—weight, 20 ozs. The wall of the left ventricle had an average thickness of eight lines; that of the right was also thickened, but the increase of its substance was in a great measure due to hypertrophy of the reticulated and fleshy bundles within, which were unusually prominent and distinct. The ventricles were not distended, though both contained large decolorised clots. The auricles were dilated and distended by loose clots. In the aorta and pulmonary artery there were insignificant atheromatous deposits; the sigmoid valves were normal. The left auriculo-ventricular communication admitted only the little finger; viewed from the auricle, it formed a rigid calcareous ring. The leaves of the valve were opaque, thickened, and rigid, as were also the tendinous cords.

The liver was enlarged and congested. The spleen was of fleshy consistence, and weighed 18 ozs. The kidneys were congested.

Remarks.—The chief value of this case arises from the fact that it was under observation for a long period; and the progress of the disease was carefully noted. It presents a very close resemblance to the preceding case; and appears to confirm the supposition that hypertrophy has a powerful counteracting influence upon the obstruction arising from the valvular disease.

In connection with the great degree of hypertrophy in these cases (X. and XI.), which to such an extent involved the right ventricle in both, it is to be remarked that both were liable to paroxysmal attacks of great severity; that these appeared very amen-

able to treatment, and an interval of very complete cessation of cardiac distress occurred; that in both the duration of the symptoms was great—in one above two years, in the other above five; that in both cases the death was sudden, though the evidence is imperfect that the mode of death in Glendinning's case (XI.) was by coma, as in Fyffe's (Case X.); and, lastly, we find in both an entire absence of hæmoptysis and pulmonary apoplexy. The agreement of these two cases in these particulars is scarcely more remarkable than is the contrast presented by the pulmonary hemorrhage and the uninterruptedly unfavourable progress of Forrester's case (IX.), which we hold depended mainly or altogether upon the dilatation.

The remarks made upon the physical signs in the two preceding cases are for the most part applicable to this case. It is farther to be observed, however, that this case presented a sign which was not ascertained to exist in either of the other cases. I refer to the grating vibration, or *fremitus*, perceptible at the apex. As appears from these cases, this sign is wanting in many instances; moreover, the history of the present case shows, that the sign occasionally ceases in a given case; and there are cases in which it is developed only when the action of the heart is excited. It is in many cases difficult to determine the precise rythm of the fremitus—*i. e.*, its relation to the sounds of the heart. It no doubt precedes the contraction of the ventricles, but it is difficult to separate it from the impulse.

This sign, when it exists, is significant of the contracted mitral aperture, so far as I have seen; though it is possible to conceive other physical conditions of the heart capable of inducing a fremitus in this situation. I have never observed it in the incompetent state of the mitral valve. In the same way, I have been in the habit of connecting the *fremitus*, which sometimes exists at the base of the heart, with pericarditic friction; for a distinctly appreciable fremitus in this situation scarcely occurs in other forms of disease. A fremitus at the base of the heart does exist in rare cases of incompetent aortic valves, but very rarely, and these cases are easily distinguishable. These observations have no reference to the *fremitus*, or "purring tremor," which exists in certain cases over the great vessels of the neck, and at times on the upper part of the sternum—the value of which, as a sign of disease, I am disposed to think has been overrated.

In the progress of the case, several of the physical signs and the action of the heart underwent changes, to which I merely direct attention, as they are recorded in the narrative of the case, without any attempt to explain them.

The pleurisy, which during life had been latent, and was brought to light only post-mortem, was not such as to influence the fatal result very materially; though, taken in connection with the bronchitis, it gives some ground for doubting that death occurred, as in

Case X., by coma, and renders it a plausible conjecture that exhaustion, influenced by these complications, and possibly inducing death by syncope, had occurred.

The complications caused some difficulty in the treatment; the chronic bronchitis was no doubt intimately connected with the paroxysms; it was always aggravated at the time they occurred, and was referred, for the most part, to unfavourable changes in the weather, and to exposure. The relief of the bronchitis was, then, always a leading object, and this was effected by ordinary means. In attempting to relieve the embarrassed state of the circulation by leeching, which always proved more or less effectual, it was found, at the same time, to relieve the epigastric pain and the vomiting, though, on some occasions, the latter did not yield until the further administration of bismuth, with or without Dover's powder, which always had the desired effect.

SECTION IV.

CONTRACTIONS OF THE VALVES.

In further illustration of this subject, I shall state, in a general way, the leading facts of sixteen cases, in which one or more of the orifices were contracted.

The early age at which the disease existed in many of these cases is worthy of special notice. In only one case was the patient above the age of 50; in two, the patients were about 40; in three, about 30; while in ten the symptoms of heart disease were manifested about or previous to the time of puberty. All of these ten patients were under the age of 20; five of them were under 15.

The influence of the sex cannot be inferred from so small a number of cases, but there was a preponderance of females—eleven out of sixteen.

The general progress, and the mode of death, may be stated as follows:—

Three cases are known to be still alive. They have laboured under the manifested effects of diseased heart, two of them for five years, and one for two; they are under the age of 15; they present the signs of a considerable hypertrophy; and, with rest and care, they enjoy long intervals of moderate health and comfort. Paroxysms of dyspnœa, with palpitation and angina, occur but rarely; and one of them is liable to severe attacks of catarrh, chiefly in winter, and after exposure. They are, without exception, indisposed to the activity which is usual in early life.

Of the remaining cases, three have been lost sight of, and ten are known to have proved fatal. In two of these the symptoms of the fatal attack were not directly referable to the disease of

the heart;—one died with the symptoms of chronic disease of the brain; she was exhausted by long-continued menorrhagia, which appeared to depend upon organic disease of the uterus, but which was, I believe, aggravated by the disease of the heart; the other case died, exhausted with the symptoms of phlebitis, and diffused inflammation of the cellular tissue of the neck; she had general chronic bronchitis, and advanced granular disease of the kidneys. In eight cases, the symptoms of the fatal illness were directly referable to the circulation. In three of these, death was sudden; the other five cases terminated, some of them by a rapid dissolution, some more gradually with apnœa, and the symptoms we have seen reason to associate with a preponderating dilatation. Dyspnœa existed in every case; dropsy was distinct only in three; angina pectoris was decided and troublesome only in one case. Of the cases suddenly fatal, two have been recorded. (X. and XI.) The third died by syncope; she had been delivered two days previously, and had had considerable hemorrhage; the fatal syncope was induced by rising from bed.

A review of the complications which accompanied these cases will throw additional light on the history of this form of disease; they involved chiefly the brain, lungs, or the heart itself.

Cerebral complications, so frequent in the course of diseases of the heart, occurred in only four of our sixteen cases. Two of these proved fatal, and one is known to be still alive. One of the fatal cases is reported at page 28 (Case X.), and was a well-marked example of apoplexy proving rapidly fatal. The other case, already referred to (page 34) in explaining the mode of death in these cases, was as follows:—

Case XII.—Margaret Mackay, æt. 27, of spare and delicate frame, was admitted, 5th May 1846, on account of profuse menorrhagia; she was much exhausted. She did not suffer from symptoms directly referable to the heart affection, except slight substernal pain; the signs of cardiac disease were, however, well marked.

About four weeks before death incomplete hemiplegia appeared; it was slight at first, but steadily increased, and was uninfluenced by treatment. Latterly there was superadded a weak and childish state of mind, and wandering delirium.

On dissection, no organic lesion of the brain was appreciable, the heart was very little hypertrophied, and the mitral orifice would not admit the little finger.

In this case, the general character of the symptoms was, probably, in a great measure owing to the exhausted state of the system, from the uterine hemorrhage.

In the other two cases, with cerebral complication, there were signs of extreme hypertrophy of the heart, contracted and incompetent mitral valve, and, in one, incompetent aortic valves. In the case with incompetent aortic valves, the cerebral symptoms consisted

of incomplete amaurosis, and paralytic feebleness of the extremities of the left side. This man was lost sight of after he left the hospital. The second of these two cases, known to be alive, is referred to at page 34. He is about the age of 16; he has laboured under cardiac symptoms for five or six years, but enjoys good health, though he is subject to occasional attacks of catarrh. He is a boy of great intelligence. His paralytic seizure was sudden, apoplectic, and occurred about two years ago; and the power of his limbs is much and permanently impaired. The signs of hypertrophy of the heart are well-marked.

Thoracic complications were more frequent. The existence of some of these could be determined during life; others could be ascertained to exist only after death. Chronic bronchitis existed in eight of our sixteen patients; but was troublesome only in six, and was complicated in some of these with chronic pleurisy. With one exception, the bronchitis was less remarkable for its permanence than for the acute paroxysmal attacks which occurred, usually after exposure; and it was generally accompanied by cardiac distress. In the intervals of the attacks the patients had perfect freedom from cough, and the signs of bronchitis nearly ceased, except in two cases, in which the bronchitis was more permanent. The facts of these cases do not, to my mind, prove a very manifest relation between the contracted orifices and bronchitis.

Pulmonary hemorrhage has been supposed to be intimately connected with contraction of the left auriculo-ventricular opening; but, from Cases IX., X., and XI., it appears that this state of the lungs depends rather upon dilatation. I shall not occupy space by general statements on this point; it would require for its elucidation careful observation, and precise statements. Of the nine cases examined post-mortem, pulmonary condensation from hemorrhage was wanting in four; two of these are given at pages 28 and 31; the other two presented less hypertrophy, but no very manifest dilatation.

The following statement of the cardiac lesions which co-existed with the mitral contraction, is drawn up chiefly from the state of the heart, as observed post-mortem, and from the physical signs, in so far as they afford information that may be trusted:—

The mitral valve, besides being contracted, was incompetent, in six of the nine cases which were examined anatomically; and from the signs in these and the other cases, it appeared to be incompetent in twelve of our sixteen patients.

Other valves were diseased in only two cases; and the proportion is even smaller than appears from this statement, inasmuch as in the other seven cases, which can scarcely be included in the calculation, as they were not dissected, not one appeared to be affected in any other valve. I regard this as an important fact, and in this respect these cases contrast with those in which other forms of valvular disease

existed; for in them the lesion is rarely limited to one valve; and, even if another valve is not involved, the endocardium and the aorta are usually more or less affected. The limitation of disease to one orifice suggests that possibly there is something distinctive in the nature of this disease, and that there is a peculiar obnoxiousness of the left auriculo-ventricular valve to the species of degeneration from which these contractions result: it is possible, too, that the delay and the abatement of symptoms in these cases is due to this limitation of the disease. It must not, indeed, be assumed, for there are not facts to prove, that the elementary forms of disease, from which the various valvular deformities result, are really different from each other. Nevertheless, the observed facts are remarkable, and of practical value. The early age at which the contraction of the mitral orifice existed in many of these cases, and the rarity with which they could be traced to any inflammatory or rheumatic attack, as they, almost without exception, were from the first chronic and latent, contrast with the age and origin in other valvular affections, and support the supposition, that there is a difference in the elementary forms of disease from which these lesions spring.

The two cases in which other valvular lesions co-existed occurred in very different circumstances, and yet they present some analogy; they tend to confirm the supposition of the distinct nature of the elementary pathological states in which mitral contractions originate; they were as follows:—

Case XIII.—Catherine Junor, æt. 22, was visited by me only a few days previous to death, in March 1845, along with Mr William Walker, who informed me that she had been liable to attacks of rheumatism for several years, and that symptoms of acute cardiac inflammation had repeatedly occurred. She was suffering from the most aggravated effects of obstructed circulation; and the signs of a dilated heart, and of contraction of the mitral orifice, existed.

On dissection, the aortic, as well as the mitral orifice were found exceedingly contracted. The heart was increased in bulk; there was general hypertrophy, but greatest of the left ventricle; and all the cavities were dilated. There were old-standing evidences of endo-pericarditis. Dropsy and pulmonary apoplexy existed.

The other case was also seen by me only during the last few days of life. She was visited by one of the pupils of the New Town Dispensary, and had not called in medical aid till the seventh day previous to death. In reporting these two cases I confine myself to such a general statement of the facts as will suffice to indicate their bearing on what has been said regarding the limitation of disease in this class of cases.

Case XIV.—Jane Grieve, æt. 26 (13th January 1848). There was universal purple congestion of the surface, amounting to slight cyanosis, slight tumidity of the face, and suffusion of the eyes. Her expression was that of a person under the influence of opium; she was torpid, but intelligent; the extremities were cold; pulse imperceptible. She complained of oppression of the chest, and slight cough, with watery and mucous expectoration of a prune-

juice colour; no dyspnœa. The discharges from the bowels, after enema, were semi-fluid, and tar-like. She suffered from an intolerably irritable and tender state of the skin.

She had been confined to bed for only twelve days. She had been previously liable to fits of vertigo, and occasional attacks of palpitation and dyspnœa; and her friends had, at times, observed an unusual "blueness" of the surface.

On dissection, both auriculo-ventricular openings were found contracted—the left in the usual manner; the right by a *membranous curtain*, which was spread across the aperture, and superseded the ordinary valvular structure; it had a perforation near its centre, about the size of a quill, which, with one other still smaller opening, constituted the communication between the auricle and ventricle. The usual muscular columns, and the tendinous cords of the tricuspid valve, were wanting; and there were extended, between the muscular wall of the ventricle and the above-mentioned membranous substitute for the valve, four or five thread-like tendons, from a half-inch to an inch-and-a-half long. These tendons appeared fitted to drag the membranous expansion, which occupied the aperture, towards the cavity of the ventricle during its systole—thus constituting a more decidedly valvular structure. The aortic aperture also was contracted by the adhesion of the contiguous margins of the sigmoid valves. Hypertrophy was manifest, though its degree could not be precisely determined. Dilatation of the ventricles was not well marked. The auricles were large and distended. Neither dropsy nor pulmonary apoplexy existed.

In the former of these cases (XIII.), the extension of disease to a second orifice was due to the extent of the endo-cardial inflammation, which may be explained by the rheumatic taint; at all events, the facts must be taken in connection. We have seen that rheumatic endocarditis is rarely the origin of these contractions; and in this solitary case in which it was, we find other orifices implicated, rendering it all the more probable that the form of disease which has been seen to limit itself to one orifice, and to have no relation to manifested rheumatism, is different.

The latter case (XIV.) is the only instance I have met with of contraction of the right auriculo-ventricular aperture; and the only case I have seen recorded is by Dr Hope. In his case, as in our patient Grieve, both auriculo-ventricular apertures were contracted; in his patient, however, as appears from his description, the contraction on both sides of the heart was due to degeneration of the tissues. This, certainly, was not the cause of the obstruction on the right side in the case of Grieve (XIV.); for the tissues presented the glistening fibrous and membranous aspect of the healthy structure; and conveyed to my mind the idea, that the state of the parts was a congenital malformation.

As it was impossible to determine precisely the degree of hypertrophy and dilatation in most of these (16) cases, I shall not examine them minutely with reference to the influence of these lesions. It may be asserted as a general fact, that hypertrophy, or increase of the muscular substance, existed in every case; though the weight of the heart in some cases did not exceed 12 oz.; we have seen in one case that it attained to 20 oz. (Case XI.) I do not think the weight of the heart exceeded 16 oz. in any other case. If, however, the youth

of many of the patients be remembered, this must be regarded as a high average weight. In the cases which struggled longest against the disease, and which had intervals of the most complete abatement of distress, the hypertrophy was most decided. Three cases, known to be still alive, present the signs of hypertrophy; and these patients have laboured under the manifested effects and signs of mitral contraction for from three to six years.

If dilatation be understood to signify merely increased capacity, then these cases were, without exception, so affected. If, on the other hand, it be understood, as the word is employed in this paper, to signify capacity increased disproportionately to the hypertrophy, and involving embarrassment of the heart, then dilatation was less invariably present. The left auricle was in every case dilated, as it always is in this form of disease. It usually contained firmly adherent decolourised coagula, which in two instances had the appearance of organized fibrinous polypi, which were pyriform, about three inches in length, and attached by their pedicle within the auricular appendage; they were hollow, and filled with a pus-like fluid, which, however, was not organized, and contained amorphous granules in such number as gave the fluid its creamy consistence. Dilatation of the right auricle, so remarkable in Case XI., did not exist to the same degree in any other case.

It is believed that there is a tendency to dilatation of the right ventricle in cases of mitral contraction; and it appears that this is, in a degree, true. I think, however, that the frequency, as well as the degree, of this dilatation, has been exaggerated; and that dilatation of the left ventricle generally co-exists. A mistaken impression has probably arisen from the fact, that the existing dilatation of the left ventricle is more or less masked and screened from observation, by the greater increase of muscular substance, which ordinarily accompanies its dilatation, than in that of the right ventricle. Cases IX. and X. are examples of the very highest degrees of dilatation on the one hand, and of hypertrophy on the other, of the right ventricle; and in none of the other cases did there exist the same contrast in the state of the ventricles; but even in Case IX., let it be borne in mind, the dilatation had seriously implicated the left ventricle. Dilatation of both ventricles existed in all the cases which had been characterised by the most decided effects of an obstructed circulation.

These cases generally corroborate what has been said of the physical signs of this form of disease. The comparative smallness of the radial pulse was invariable. Fremitus, or grating tremor, at the apex, was ascertained in eleven of the sixteen cases—it probably existed in a larger proportion; it was observed to cease at times, and in one case it disappeared altogether; it was in most instances perceptibly antecedent to the ventricular systole. This sign was not present in the case (XIV.) in which both auriculo-ventricular apertures were contracted.

Pregnancy and labour are variously influenced by disease of the heart; and the favourable or unfavourable result probably depends upon distinct physical conditions of the organ. The only fatal cases I have met with had contraction of the mitral orifice: I, therefore, introduce the following one here—not, however, supposing that this state of the valve has any peculiar influence. The other case is referred to at page 35.

CASE XV.—*Contraction of the Mitral Orifice; Labour; Hæmoptysis; Sudden Death.*

Mrs Abbot, æt. 26, a spare and delicate woman, the mother of two children, was visited by me on the 14th November 1849, with Dr Buchanan. She was in the fifth month of her third pregnancy, and at the time comparatively free from suffering, though the slightest exertion caused dyspnœa and palpitation; and a protracted attack of profuse hæmoptysis had just ceased.

History.—Symptoms of cardiac disease first attracted attention during the preceding pregnancy—nearly two years previously. Since that time she had been delicate, liable to dyspnœa, palpitation, cough with scanty expectoration, and a sense of epigastric weight and oppression; hæmoptysis had been frequent, and at times profuse of late. Dr Buchanan attended her in her last labour in March 1848, which was natural. She had never had dropsy, so far as she was aware, nor any acute disease; no rheumatism.

Physical Signs.—The radial pulse was small, contrasting with the impulse of the heart, which, however, was neither forcible nor extended; the *apex cordis* beat in the usual situation, and its impulse was *preceded* by a grating *fremitus* or thrilling tremor. The præcordial space dull on percussion, extended from the third left rib four and a-half inches downwards, and from the mesial line three inches leftwards. The sounds were difficult to analyse, they were free from murmur at the base, and a harsh prolonged murmur existed at the apex. Hepatic dulness on percussion extended to the epigastric space.

Progress.—Labour occurred on the 6th February, and progressed favourably, except that she coughed a good deal; she was delivered about one in the morning of the 7th, and appeared well and comfortable. Dr Buchanan remained with her for two hours. About four in the morning sudden and profuse hæmoptysis occurred, and she died almost immediately. Dr Buchanan was not sent for, and on the following morning he could only learn that she started up in bed when the attack occurred, and that there had been violent cough and excessive dyspnœa. From November till the date of labour she had not been confined to bed, trifling hæmoptysis had recurred again and again, and she had been unfit for exertion. The child, though premature, was vigorous, and is well.

Remarks.—In the absence of anatomical demonstration, it must be assumed that pulmonary apoplexy was the immediate cause of death; that this arose in consequence of dilatation of the ventricles of the heart; and that the primary disease was contraction of the mitral orifice.

It appears that disease of the heart existed at the time of her previous labour; and accoucheurs are familiar with a favourable result of labour, notwithstanding the existence of heart disease. The question consequently is, what difference exists between the favourable and unfavourable cases? I believe that the progress of Mrs Abbot's case, during the last two years, was due to a slowly advancing dilatation; and that, in favourable cases, preponderating hypertrophy will be

found to exist. Accidents, perhaps, sometimes arise along with minor degrees of dilatation after labour, just as they appear at times to arise in consequence of accidental inflammatory attacks (see pages 46 and 47); and it will not be always possible to show how far the result is influenced by the labour.

In Dr Buchanan's patient, Mrs Abbot, as well as in my other case, death was delayed some time after labour, and resulted in a manner not unfrequent in the diseases of the heart,—by *asphyxia* in the former, and by *syncope* in the latter; *both* of which accidents we have seen reason to associate with dilatation. In a case recorded by Dr M'Gowan, on the other hand, death occurred instantly after delivery; and this may be explained by the extreme degree of dilatation, and the more advanced effects of the heart disease, in connection with a morbid state of other organs.[1]

Important practical questions, on which we are very imperfectly informed, are involved in the management of such cases. We have not at present to do with their management during pregnancy, but in and after labour. In such an attack as proved fatal in Mrs Abbot's case, were the opportunity timeously afforded, I would be disposed to employ general bleeding, with the view of more speedily disembarrassing the action of the heart, although general blood-letting is ordinarily contra-indicated by dilatation; and the desired relief will be more safely obtained by early and repeated leeching, immersion of the extremities in hot stimulating liquids, &c.

In regard to the management of labour, Dr Simpson expresses a general opinion, that "the existence of disease of the heart is an indication for earlier instrumental interference." This opinion was suggested by Dr M'Gowan's case, and it appears a safe, perhaps a necessary, practice; but I think it is not an unfounded fear, that earlier, unless very much easier, delivery, would not have obviated fatal syncope where disease of the heart was so advanced. Besides, the many prosperous cases which occur show that interference is not always necessary; and in the case of Mrs Abbot, as well as in the one referred to at page 31, the fatal accidents were but indirectly connected with labour, and could have been obviated, if at all, only by treatment subsequent to delivery. In making these observations, I have had in view rather to suggest farther inquiry, than to offer

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[1] As Dr M'Gowan's case is important, I subjoin a condensed account of it,—abstracted from the proceedings of the Edinburgh Obstetric Society, January 20th, 1849.

Mrs Baker, æt. 21, was visited by Dr M. on the 16th June 1845. She had spurious labour pains, dyspnœa, pain of the left side, and dropsy; she was bled from the arm and cupped. Labour occurred on the 20th; it lasted six hours; the child was still-born, and she instantly expired. On *post-mortem* examination, the heart was enlarged and distended; the right ventricle was very thin and dilated; the aortic orifice was contracted. The left lung was extensively condensed.

rules for the discrimination and treatment of this class of cases. Our knowledge of the reciprocal influences of pregnancy, labour, and the diseases of the heart, is very inexact; and there is a want of carefully observed cases.

SECTION V.

INCOMPETENCE OF THE VALVES.

The influence of hypertrophy and dilatation admits of still farther illustration from other forms of cardiac disease;—cases of which are more familiarly met with than some of those already recorded. Incompetence of the valves is one of the most usual results of acute or chronic disease of the endocardium; but it is a purely physical and accidental effect; and it never proves fatal without the development of hypertrophy or dilatation of the heart.—The exact relation of hypertrophy or dilatation to valvular incompetence, is not well made out; but this question involves speculative considerations which would here be out of place.

In recent acute endocarditis, causing valvular incompetence, the circulation is in many cases less disturbed than in cases in which the valves remain competent, showing that regurgitation is not of itself sufficient to induce a seriously obstructed circulation. But this is still more remarkable in some chronic cases, in which there exists unmistakeable evidence of valvular incompetence without obstruction, or even embarrassment of the circulation. This is well seen in the two succeeding cases, in which no signs of dilatation existed, and but slight hypertrophy had occurred.

CASE XVI.—*Incompetence of the Aortic Valves; Chronic Rheumatism.*

James Coggans, æt. 32, a robust labourer, applied to me in April 1845, for relief of unceasing pain and tenderness in the left temple. He had none of the usual manifestations of rheumatism, but said he had had "pains," and had taken mercury.

On examination, a decided bellows murmur accompanied the second sound at the base of the heart. The first sound was muffled, and the space dull on percussion was extended, but little.

He was under observation for two months, during which the signs continued without change, and he had occasional sense of palpitation.

CASE XVII.—*Incompetence of the Aortic Valves.*

David Macallum, æt. 22, a baker, was admitted into the Royal Infirmary on the 11th December 1849. He was spare and slender, but not of unhealthy appearance, temperate, liable to palpitation on exertion; he had slight cough, occasional dyspnœa, and alarming dreams. He said he had been unfit for regular work for four years, and ascribed his complaints to the effort of lifting and carrying burdens.

On examination, he breathed easily, and was free of pain; his chest was capacious and well-formed; the heart's action was excited, with visible pulsation in the

fourth and fifth intercostal spaces. The pulse was quick in its action, slightly visible, and compressible ; the præcordial space dull on percussion, measured three inches in the vertical, and in the transverse direction ; there was a well-marked bellows murmur with the second sound at the base of the heart.

He remained in hospital till the 17th January ; he was languid and indisposed for exertion, and little benefited by treatment.

The proof afforded by these cases is not complete ; still they show that valvular incompetence, even of considerable standing, is not productive of an obstructed circulation. The signs of dilatation were not marked in either case ; in Macallum I am disposed to associate the cardiac symptoms, and the increased *transverse* diameter of the præcordial space, with commencing dilatation and impaired muscular development.

The following case, one of a class frequently met with, shows how entirely obstruction of the circulation is wanting in hypertrophy of the heart without dilatation.

CASE XVIII.—*Cerebral Apoplexy ; Hypertrophy of the Ventricles of the Heart ; Disease of the Aorta.*

——— Thomson, æt. 50, a robust but intemperate man, was attacked, while at work, with apoplexy ; there was no treatment for three days, and when brought to the hospital, there was slight febrile action, stupor, and paralysis of the right side. Within a year he had had repeated attacks of transient insensibility. He died on the eighth day of the attack.

On dissection, the left hemisphere of the brain contained in its substance, and in the ventricle, a coagulum of blood, probably eight ounces in weight. The arteries were diseased, though not calcareous. The heart weighed fourteen ounces, and had the normal conical form. The increase of the muscular substance was chiefly in the left ventricle, the form and capacity of which were normal. The valves of the heart were competent. In the aorta, near its orifice, there was a dense calcareous excrescence about the size of a filbert ; and the surface of the artery presented many spots of atheromatous degeneration.

This case was equally remarkable for the want of dilatation of the heart and obstructed circulation ; and for the accidents which originated in the hypertrophy, excited, it may be, by intemperance.

Besides the preceding cases (XVI., XVII., and XVIII.), in which we have seen that the secondary effects of cardiac diseases, which arise from an obstructed circulation, were wanting in the absence of dilatation, an endless number of cases might be cited, in which the gravest effects of obstructed circulation resulted in connection with dilated hypertrophy of the heart along with valvular incompetence ; in fact, it is true of these cases, as we have seen it is in other forms of cardiac disease, that a permanently and fatally obstructed circulation does not occur except in connection with disproportionate dilatation. In illustration of this statement, we may refer to cases I., II., V., VIII., XXI., and XXII. ; and from the following case, it appears that a complication so formidable as adhesion of the pericardium with valvular incompetence, is of itself insufficient to cause obstruction of the circulation, except through the medium of dilatation of the ventricles.

CASE XIX.—*Incompetence of the Mitral Valve; Adhesion of the Pericardium; Hypertrophy; Dilatation; Death unexpected.*

John Strachan, æt. 49, a sailor, was admitted February 18th 1845, with the symptoms of obstructed circulation, orthopnœa, œdema, and scanty urine.

History.—His health had been good till within eight weeks, and he had been at work till within four. His symptoms supervened gradually, and latterly he had been much distressed by pain of the belly and exhausting bowel complaint, alternating with constipation.

Physical Signs.—The action of the heart was tumultuous, and the synchronism of the radial pulse, with the impulse, was imperfect; the impulse was diffused, with a "jogging" movement, to the epigastrium. The præcordial space dull on percussion, measured four inches in either direction. The sounds of the heart were indistinct,—the first accompanied by a soft bellows murmur, which was most distinct at the *apex*. The signs of general bronchitis were also present.

Progress.—Treatment afforded only temporary relief; and he was not subsequently able to leave bed. Latterly, dropsy increased greatly, and he died unexpectedly on the 3d March.

Post-mortem Examination.—Firm adhesion of the pericardium existed posteriorly near the base; elsewhere the surfaces were free. The heart weighed twenty-eight ounces, was large, globular, and the capacity of both ventricles was much increased. The tendinous cords and the leaves of the mitral valve were thickened, rigid, and opaque. The aorta was slightly dilated, and its inner surface was rough from atheromatous and calcareous deposit. The lungs, and the abdominal viscera, were loaded with blood, and the different serous cavities contained a little dark-coloured serum.

It cannot be supposed that the pericardial adhesion, the endocardial disease, and the hypertrophy, had occurred so recently as, from the history of the case, the symptoms appear to have arisen; and, though it is impossible to prove that dilatation became disproportionate only at the date of his first distress, this is not an unreasonable conjecture, and to that dilatation I would ascribe the train of symptoms which occurred during his illness. The case illustrates, in several respects, the relations of hypertrophy and dilatation;—we see extreme dilatation superadded to the highest degree of hypertrophy; and, while it is evident that organic disease of the heart must have long existed, there was up to a late date a complete immunity from the symptoms of cardiac embarrassment. This circumstance was probably due to the progressive hypertrophy of the heart,—at all events, though the connection of these is not capable of exact demonstration, their co-existence must not be lost sight of. And in this case we see a farther illustration of what is demonstrated in case XVIII., that hypertrophy, even of the highest degree, does not induce obstruction of the circulation, unless dilatation has been superadded.

Adhesion of the pericardium has been said to manifest itself by a peculiarity of the impulse, which certainly existed in Case XIX.—the impulse communicated to the epigastrium, with an undulating and "jogging" movement. This state of the impulse accompanies diseases of the heart in which no pericardial adhesion exists; and, in

the case of Strachan (XIX.), the dilated hypertrophy and the valvular disease must have influenced the character of the impulse; and besides, how frequently do we find that adherent pericardium only reveals itself post mortem? My own opinion is, that the manifestations of heart disease will not arise, in connection with adherent pericardium, so long as hypertrophy or dilatation have not occurred, as was observed in the following case :—

A vigorous young man was admitted into hospital, labouring under an attack of malignant confluent small-pox. His previous health had been good. He sank on the 10th or 11th day. The pustules had extended to the mucous surfaces of the air-tubes and gullet ; the *pericardial surfaces* were adherent by firm cellular connections over two-thirds of their surfaces.

The effects which we have now seen to be so intimately connected with dilatation, have been supposed to arise from a disorganised state of the muscular substance of the heart. The following case throws doubt on this supposition :—

CASE XX.—*Incompetence of the Mitral Valve; Hypertrophy ; Dilatation ; Fatty Degeneration ; Death by Exhaustion.*

James Chalmers, æt. 20, was admitted on the 7th January, 1847. He was sallow, bloated ; and had been intemperate. He had been suddenly attacked, four days previously, by pain of the left side, with febrile symptoms. He had had cough for above two months ; and his general health had been long deliciate. Pulse was 100, and irregular ; bowels torpid, and the abdomen tumid and semi-fluctuating.

Physical Signs.—The impulse of the heart was obscure and undefined ; the extent of the præcordial space dull on percussion was increased. The first sound was superseded by a bellows murmur, most marked at the *apex*. In both dorsal regions the murmur of inspiration was masked by a harsh mucocrepitating rattle.

Progress.—Steadily unfavourable. Urine was albuminous, constipation obstinate ; he was much distressed by sickness and pyrosis ; cough did not abate ; and he sank on the 19th.

Post-mortem Examination.—Volume of flesh was small. The lungs presented traces of general bronchitis. The heart was increased in bulk, and its walls were thickened ; the muscular substance was extensively, and in its entire thickness, converted into a pure adipose tissue. The liver was large, and fatty. The kidneys were grey, and granular.

The absence of a manifestly obstructed circulation in the preceding case is remarkable ; and the previously languid state of his health was not more evidently connected with the state of the heart than with the disease of the liver or kidney.

Hypertrophy is at times in a remarkable manner limited to one or other of the ventricles, and this may occur without apparent cause. On the other hand, it appears to arise occasionally in connection with particular forms of valvular disease. There are no cases in which a *limited* or *excessive* hypertrophy is more remarkable than in incompetence of the aortic valves,—as was well shown in Case XXII. ; but it sometimes exists without valvular disease.

To this state of the left ventricle may be traced the occurrence of cerebral and other hemorrhages from the arterial system, and the greatly increased vertical diameter of the præcordial space; and possibly the displacement of the *apex* to the sixth intercostal space is due to the same cause—on this point I cannot speak positively, as I have not noted the precise state of the ventricle in the cases in which the impulse of the apex was displaced. Excessive hypertrophy of one ventricle does not prevent its subsequent dilatation; and the other ventricle usually becomes involved, unless a conclusion is put to the case by some of those accidents to which the subjects of hypertrophy are liable.

In many cases in which valvular disease had long existed, symptoms of obstructed circulation arose only a short time before death; and the progress was steadily and rapidly unfavourable. This fact involves the consideration, already referred to, of the influence of hypertrophy in delaying the embarrassment of the heart's action, until disproportionate dilatation is developed. In cases where there is reason to believe that heart-disease of old standing had existed, without cardiac embarrassment, I have always found hypertrophy of both ventricles, sometimes in very high degree (Case XIX.); though the degree of the hypertrophy did not always bear a precise relation to the absence or delay of obstructed circulation—a minor degree of hypertrophy proving sufficient, in the absence of dilatation, to maintain the equilibrium of the circulation. The necessity of dilatation of the heart, usually involving both ventricles, in order to establish obstruction of the general circulation, is evident from the facts of many cases; but it is also true that a greatly increased capacity of the ventricles in not a few cases fails to occasion obstruction, and in such cases may be frequently seen the influence of acute local inflammations, or other accidental causes, in developing the effects of dilatation, which, in the previously undisturbed state of the system, had not manifested any of its graver effects.

Case XXI.—*Incompetence of the Mitral Valve; Hypertrophy of the Heart; Dilatation; Pleuro-Pneumonia; Jaundice*; *Death by Apnœa.*

Colin Matheson, æt. 36, a shipwright, admitted 22d February 1847. Of temperate habits. He had a bloated countenance; general anasarca; and he suffered much from dyspnœa; cough, with hæmoptysis and sickness. Pulse was 120, and irregular; urine scanty, and slightly albuminous.

History.—He had had good health till three months previously; and had suffered chiefly from cough, with expectoration, and deep-seated epigastric uneasiness. Swelling of the surface had appeared within a fortnight; hæmoptysis had occurred, for the first time, within three days.

Physical Signs.—Heart's action was tumultuous; impulse diffused, and communicated to the epigastrium; the arterial pulse and impulse of the heart were not synchronous. The sounds of the heart were, in the first instance, confused and faint; subsequently, when the action moderated, a murmur with the first sound, at the apex, was distinct. In both dorsal regions percussion was impaired; the sound of respiration harsh and feeble on the right.

Progress and Treatment.—He derived immediate benefit from leeching, purgative medicine, and repeated doses of opium and ipecacuan powder. Hæmoptysis and dyspnœa did not altogether subside. On the 27th, aggravation of his distress occurred suddenly; with dyspnœa, cough, and vomiting; and jaundice supervened. He sank on the 7th March.

Post-mortem Examination.—Volume of flesh was good; there was jaundice, and general dropsy. The right pleura contained a gallon of turbid serum, and much recent lymph. This lung was compressed; condensed in its lower lobe, from pneumonia; and the middle as well as the inferior lobe contained masses of hemorrhagic condensation. The heart weighed 27 ozs.; it was globular, dilated, and distended. The leaves of the mitral valve were thickened and rigid, as were also the aortic valves—though competent. The endocardium and tendinous cords of the left ventricle, and the interior of the aorta, were atheromatous and rough. The liver was large, and congested. The kidneys were grey, and granular.

In the meantime I shall only direct attention to the recent rise and rapid progress of symptoms in this case, the degree of hypertrophy, and the state of the right lung.

Case XXII.—*Incompetence of the Aortic Valves; Dilatation of the Heart; Hypertrophy; Pneumonia; Death by Apnœa.*

Margaret Forbes, æt. 36, a washerwoman, of full but lax development, admitted 16th March 1848. Much distressed by dyspnœa, pectoral oppression, &c.; no dropsy.

History.—Her health had not attracted her attention till three weeks previously. She was then suddenly, and after exposure, attacked with cough, breathlessness, and hæmoptysis. She had previously experienced occasional disinclination for exertion, and difficult breathing.

Physical Signs.—The pulse was 120, small, and not abnormally posterior to the impulse of the heart, which was diffused and obscure. The præcordial space was extended, and at the base of the heart both sounds were superseded by bellows murmurs. Bronchitic wheezing and mucous rattle accompanied the sounds of respiration.

Progress and Treatment.—She was temporarily relieved after local bleeding; diuretics were ineffectual. Urine became slightly albuminous. Insufferable pectoral anguish recurred with dyspnœa, and fits of approaching syncope. On the 7th of April, after an interval of some days, the symptoms recurred, and she died exhausted on the following day.

Post-mortem Examination.—The heart weighed 17 ozs. Hypertrophy existed chiefly in the left ventricle; both ventricles were dilated. The aortic valves were puckered, and incompetent; the orifice small; and the ascending aorta atheromatous and dilated. The left lung presented the condensation of pneumonia in its lower lobe, both lungs were loaded with blood, and the air-tubes contained much mucus. The liver was fatty, and congested. The kidneys were pale, and fatty.

In these cases we see the grave effects which may result from inflammatory attacks occurring in the train of chronic cardiac disease. In this there is nothing peculiar to heart disease; for we see the same untoward results, from apparently slight causes, in connection with other organic lesions, which may have been previously unmanifested.

There is great variety in the manner in which pulmonary complication is associated with disease of the heart; its influence, however, is, under all circumstances, most formidable, and it demands direct and active treatment. It is not always possible to determine whether the

pulmonary lesion originated the existing attack, or whether it arose *late*, and as a sequence of the disordered circulation; but this does not involve any practical difficulty. The complication never exists in a simple form; hence the uncertainty of its indications during life. Bronchitis is rarely wanting, and it has an admitted relation to the disease of the heart as an effect; pleurisy and pneumonia usually co-exist, and the signs of the former more or less mask those of the latter. It is in such cases that blistering is serviceable—subduing pleurisy as well as bronchitis. The chronic affections of the heart itself are not benefited by counter-irritation; but relief of the respiratory function, impeded from pulmonic complication, by counter-irritation or otherwise, goes far to diminish obstruction of the circulation.

Decided jaundice does not arise frequently in the progress of diseases of the heart; and, considering the direct influence of the obstructed circulation on the liver in all the forms of disease of the heart, this is remarkable. I do not remember to have met with it, except in Cases I. and XXI.; in both of these cases it occurred shortly before death, and no treatment had any effect upon it.

In former cases it has been shown, that the condition of the right ventricle exerted a powerful influence on the state of the circulation (see Cases IX. and X.); and it has been argued that a patent state of the right auriculo-ventricular communication is a chief cause of the obstructed state of the circulation, which I have been led to connect rather with general dilatation, possibly of *one*, probably of *both*, ventricles. It has been almost established, that a degree of incompetence of the tricuspid valve is not inconsistent with health; and I believe that the higher degrees of widening of this aperture arise only in connection with dilatation of the right ventricle; and, moreover, there ordinarily co-exists more or less dilatation of the left ventricle. It is impossible to attribute the obstruction of the circulation to one of these lesions to the exclusion of the others; but, if one has a primary importance, I believe it is the dilatation of the ventricles.

Valvular disease of the right side of the heart is admittedly rare; I believe, nevertheless, that its frequency has been overrated. Case XIV. is the only one I have met with in which its existence was demonstrated. In another case the physical signs were such as to indicate incompetence of the pulmonic valves; but, in the absence of anatomical demonstration in that case, as well as in all those I have seen described, I cannot affirm that the lesion existed, and the general symptoms in the case were not important.

The physical signs of valvular incompetence require little notice—they are for the most part well understood, though the value of some has been over-estimated. Confusion has arisen from importance being attached to the character or tone of the endocardial murmurs; their true diagnostic value depends upon two circumstances—the *position* in which they are most distinctly audible, and their relation to the

rythm of the heart's action. It matters not whether the endocardial murmur be musical, grating, or soft and blowing, with the exception already indicated in contraction of the mitral orifice, in which the characteristic murmur appears to have a *harsh grating* character—(see Cases X. and XI., pages 28-31). In illustration of this point, I shall only refer to two cases which have been brought under my notice; in both, incompetence of the aortic valves was attended by a murmur so loud and shrill that it was audible at a distance of several inches from the surface. But the nature or seat of the disease in these cases was determined solely by the *position* and *rythm* of the murmur, which existed, as in all such cases, at the *base* of the heart, with the *second sound*. The musical character or loudness of the murmur was connected with the intensity of the vibration, caused by the regurgitant current, which was accompanied in both cases by a vibrating *fremitus*—a sign not usually observed in aortic incompetence.

The various changes which the pulse undergoes are more intimately related to changes of the muscular substance, than to valvular lesions. There are conditions of the pulse which depend only upon valvular disease; but these may be limited to two—the small pulse of mitral or aortic contraction, and the quick diastolic pulse of aortic regurgitation.

The *visible* pulse is not always an abnormal condition; but, even in its higher degrees, it has no relation to valvular disease. *Irregularity* has been regarded as significant of mitral incompetence; but it frequently exists in cases where there is no valvular disease; and it occurs with mitral incompetence, in such circumstances as lead me to believe that it arises only in consequence of superadded dilatation.

Want of *synchronism* of the arterial pulse with the heart-beat has a doubtful connection with valvular disease; it does not occur, except in conjunction with more or less dilatation; and I have met with it altogether apart from valvular disease.

Delay of the radial pulse has been stated to depend upon incompetence of the aortic valves; I do not think such is the fact. The radial pulse is naturally more or less posterior to the heart's impulse; and the interval may vary within the limits of health; besides, from the very nature of the thing, it is difficult or impossible to affirm in a particular case that the *degree* of delay is abnormal. From my own observations, I am satisfied that an appreciable increase in the delay of the arterial pulse is very rare; and it does not arise in such a proportion of cases of aortic incompetence as to indicate any relation between them.

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